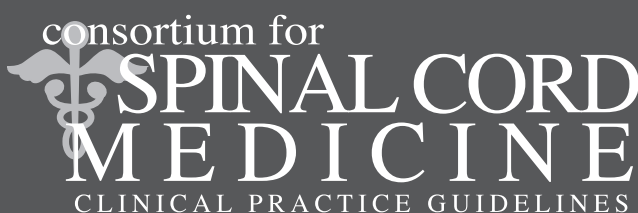


Early Acute Management in Adults with Spinal Cord Injury:

**A Clinical Practice Guideline
for Health-Care Professionals**



Administrative and financial support provided by
Paralyzed Veterans of America

Consortium for Spinal Cord Medicine Member Organizations

American Academy of Orthopaedic Surgeons
American Academy of Physical Medicine and Rehabilitation
American Association of Neurological Surgeons
American Association of Spinal Cord Injury Nurses
American Association of Spinal Cord Injury Psychologists and Social Workers
American College of Emergency Physicians
American Congress of Rehabilitation Medicine
American Occupational Therapy Association
American Paraplegia Society
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Society of Critical Care Medicine
U. S. Department of Veterans Affairs
United Spinal Association

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This guideline has been prepared based on the scientific and professional information available in 2006. Users of this guideline should periodically review this material to ensure that the advice herein is consistent with current reasonable clinical practice. The websites noted in this document were current at the time of publication; however, because web addresses and the information contained therein change frequently, the reader is encouraged to ensure their accuracy and relevance.

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The Consortium for Spinal Cord Medicine has published the following guidelines to date:

Bladder Management for Adults with Spinal Cord Injury

Preservation of Upper Limb Functioning Following Spinal Cord Injury

Respiratory Management Following Spinal Cord Injury

Acute Management of Autonomic Dysreflexia: Individuals with Spinal Cord Injury Presenting to Health-Care Facilities, 2nd edition

Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury

Outcomes Following Traumatic Spinal Cord Injury

Prevention of Thromboembolism in Spinal Cord Injury, 2nd edition

Neurogenic Bowel Management in Adults with Spinal Cord Injury

Depression Following Spinal Cord Injury

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Foreword

Spinal Cord Injury: The First 72 hours

In the Introduction to his 1982 text *Early Management of Acute Spinal Cord Injury*, Charles Tator stated, “The early management of a patient with an acute spinal cord injury is one of the most difficult tasks in trauma cases. During the first few days after an acute cord injury, every physician, nurse, or paramedical person coming in contact with a cord injured patient bears a major responsibility” (Tator, 1982). He added that the “final outcome of a spinal cord injury depends upon the accuracy, adequacy, and speed of first aid management, diagnosis, and treatment within the first few hours” (Tator, 1990). Our efforts to prevent spinal cord injury have borne only modest success, and the condition remains as much of a threat to life and health as ever. However, our understanding of the physiology involved is improving, and evidence is slowly accumulating to guide us in our management decisions. Is the evidence sufficient to support us in our care of the newly cord injured person?

Sackett et al. (2000) delineate two distinct components of guidelines for clinical practice: first, the summary of the evidence upon which the guidelines are based, and second, the detailed instructions or recommendations for applying that evidence to our patients.

Following the protocols developed by the Consortium for Spinal Cord Medicine, our panel has reviewed the evidence pertaining to care of the patient with a new spinal cord injury, focusing on the first 72 hours of injury. This care is in the hands of many people, from the prehospital providers first on the scene, to the spine surgeon providing definitive care, to the physiatrist initiating rehabilitation (which should begin in the intensive care unit). Along the way, the team will include emergency physicians, radiologists, respiratory specialists, intensivists, and many other clinical personnel who spend varying percentages of their workweek with persons with new spinal cord injuries. This team needs to know the specific issues—and the related scientific evidence—relevant to this group. We hope that all those involved in early care will find this guideline a helpful reference.

Throughout, we have adhered to the terminology employed in the *International Standards for Neurological Classification of Spinal Cord Injury* (Marino, 2002). Thus, tetraplegia (not quadriplegia) is the preferred term for a spinal cord deficit affecting the upper extremity and paraplegia for that affecting only the lower part of the body excluding the upper extremities.

The panel members have carefully considered the best evidence available in making recommendations for clinical care. We believe that these are standards to aim for, recognizing that resources may limit our reach. Our recommendations will stand for a while, until our understanding evolves based on new evidence. This evolution is part of the excitement of medicine and rehabilitation science. It remains for the reader to take our recommendations into consideration as newer evidence becomes available. We may also see great changes over the next few years as translational research suggests the careful deployment of interventions shown to be of value in the lab, which must be responsibly evaluated in humans in a controlled setting before being widely used.

Peter C. Wing, MB, MSc, FRCSC
Panel Chair

Preface

As chair of the Steering Committee of the Consortium for Spinal Cord Medicine, it is a distinct pleasure for me to introduce our 10th clinical practice guideline, *Early Acute Management in Adults with Spinal Cord Injury*. This guideline was developed by an expert panel encompassing the myriad disciplines that care for a person from the time of injury through the critical first few days. In fact, it is those first few days after injury that are the most crucial in terms of survival, neuroprotection, prevention of secondary complications, and psychosocial adjustment to the drastic change in life circumstance. Survival and preservation of neurological function are dependent on an effective system of care that includes prehospital management, trauma centers, and spinal cord injury centers. Neuroprotection efforts begin at the scene of the injury with proper immobilization of the spine and cardiorespiratory stabilization, followed by accurate clinical and radiographic evaluation and spinal stabilization. At this time, there is insufficient evidence to definitively support neuroprotection using pharmacological agents and other modalities such as cooling; however, this is an area where future research is expected to yield rich results in terms of improved neurological outcomes following an injury to the spinal cord.

During the first few days when life-saving interventions dominate the care of the spinal cord injured individual, efforts at preventing secondary complications become vital. Certain complications, such as venous thromboembolism, primarily occur during the acute period; others, such as pressure ulcers and respiratory and urological complications, may first appear during the acute period but may also be long-term complications. Preventive measures administered during the acute phase may have lifelong benefits. On top of all this, the emotional consequences of a spinal cord injury are immense and affect the injured individual as well as the family. Addressing the psychosocial needs of all concerned will be a continuous process, but it is crucial to begin during the first few days after injury.

On behalf of the consortium steering committee, I want to acknowledge Dr. Peter C. Wing's expert, passionate, and committed leadership of our distinguished guideline development panel. Each distinguished panel member brought to the guideline development process an immense amount of energy and dedication for the care of people with spinal cord injury (SCI). Special thanks also go to representatives of the consortium's 22 member organizations, who thoughtfully and critically reviewed the draft in its various forms. Their contributions were essential to making this document one that will improve both the quality of care and the quality of life for persons with SCI.

The development of this clinical practice guideline is dependent on the exceptional administrative support and other services provided by the Paralyzed Veterans of America. The consortium is profoundly grateful to Paralyzed Veterans' Executive Committee, led by National President Randy L. Pleva, Sr., and to the Paralyzed Veterans Research and Education Department. Thomas E. Stripling, Director of Research and Education, Kim S. Nalle, Manager of Clinical Practice Guidelines, and Caryn Cohen, Associate Director, Clinical Practice Guidelines, are instrumental to all aspects of the development of these guidelines, from inception of the topic through dissemination. We could not do it without them. The Consortium is very appreciative of the Ron Shapiro Charitable Foundation for its financial support for methodological resources and the printing and distribution of this clinical practice guideline.

Lawrence C. Vogel, MD
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Acknowledgments

The Consortium for Spinal Cord Medicine spans many disciplines, and we appreciate the generous support given to the process by the many organizations represented. Particular mention must be made of the Society of Critical Care Medicine, with whom we worked closely in assembling this guideline. Many of their members are on the front line of providing care to cord injured people, and we are grateful that they elected to work with us in developing recommendations to better ensure the quality of care. The guideline is the better for their input.

We have been supported in this work by many, unnamed colleagues who have reviewed sections of the guideline and made helpful suggestions. Thank you all.

Paralyzed Veterans continues its vital role as sponsor of this series and coordinator of the development process. This guideline owes much to the experienced hands and minds of J. Paul Thomas, Thomas Stripling, Kim Nalle, and Caryn Cohen, and the panel thanks them for their persistent and constructive support.

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Summary of Recommendations

Triage Protocols and Trauma Systems of Care

Prehospital Triage

1. Develop appropriate guidelines for the evaluation and transport of patients with potential spinal cord injuries based on local resources. Identify regional trauma centers with special resources for the acute management of spinal cord injuries.

Trauma Centers

2. Transfer the patient with a spinal cord injury as soon as possible to a Level I trauma center, as defined by the American College of Surgeons or by state statute. Given local triage protocols and guidelines relating to transportation times to trauma centers, consider taking the patient directly to a Level I center if possible in preference to passing through a Level II or III center first.

Spinal Cord Injury Centers

3. Consider directing spinal cord-injured patients expeditiously to a specialized spinal cord injury center that is equipped to provide comprehensive, state-of-the-art care. Discuss pretransfer requirements with the referral center.

Spinal Stabilization during Emergency Transport and Early In-Hospital Immobilization Following Spinal Cord Injury

4. Immobilize the spine of all patients with a potential spinal injury from the scene of the injury to definitive care.
5. Emergency medical service (EMS) providers should use the following five clinical criteria to determine the potential risk of cervical spinal injury:
 - Altered mental status
 - Evidence of intoxication

- Suspected extremity fracture or distracting injury
- Focal neurological deficit
- Spinal pain or tenderness

6. EMS providers should use the combination of rigid cervical collar immobilization with supportive blocks on a backboard with straps or similar device to secure the entire spine of all patients with potential spinal injury.
7. In the emergency department, transfer the patient with a potential spinal injury as soon as possible off the backboard onto a firm padded surface while maintaining spinal alignment.
8. In cases of confirmed spinal or spinal cord injury, maintain spine immobilization until definitive treatment.
9. At the extremes of age, or in the presence of a preexisting spine deformity, provide patient care in the position of greatest comfort while maintaining immobilization.
10. Employ an adequate number of personnel during patient transfers for diagnostic studies and for repositioning to maintain the alignment of a potentially unstable spine and avoid shearing of the skin.
11. Logroll the patient with a potentially unstable spine as a unit when repositioning, turning, or preparing for transfers.
12. Consider a specialized bed for the patient with an unstable spine when prolonged immobilization is anticipated.
13. Initiate measures to prevent skin breakdown if prolonged time on a backboard is anticipated.
14. Perform a baseline skin assessment on removal of the backboard.

'ABCs' and Resuscitation

15. Provide airway and ventilatory support in patients with high tetraplegia early in the clinical course.
16. Prevent and treat hypotension.

17. Determine initial base deficit or lactate level to assess severity of shock and need for ongoing fluid resuscitation.
18. Exclude other injuries before assigning the cause of hypotension to neurogenic shock.
19. Recognize and treat neurogenic shock.
20. Monitor and treat symptomatic bradycardia.
21. Monitor and regulate temperature.

Neuroprotection

Pharmacologic Neuroprotection in Patients with Spinal Cord Injury

22. No clinical evidence exists to definitively recommend the use of any neuroprotective pharmacologic agent, including steroids, in the treatment of acute spinal cord injury in order to improve functional recovery.
23. If it has been started, stop administration of methylprednisolone as soon as possible in neurologically normal patients and in those whose prior neurologic symptoms have resolved to reduce deleterious side effects.

Diagnostic Assessments for Definitive Care and Surgical Decision Making

Clinical Neurologic Assessment for Spinal Cord Injury

24. Perform a baseline neurological assessment on any patient with suspected spinal injury or spinal cord injury (SCI) to document the presence of SCI. If neurologic deficits are consistent with SCI, determine a neurological level and the completeness of injury. Perform serial examinations as indicated to detect neurological deterioration or improvement.

Radiographic Evaluation of Patients Following Spinal Cord Injury

25. Image the entire spine in a patient with an SCI.
26. Perform magnetic resonance imaging (MRI) of the known or suspected area(s) of spinal cord injury.

Premorbid Spinal Conditions and the Extremes of Age: The Mobile and the Stiff Spine

27. In patients with SCI, be aware that bony imaging of the spinal column may be negative (i.e., "SCTWORA," or SCI without radiological abnormality).
28. In a patient with a stiff spine and midline tenderness, suspect a fracture. Consider MRI, bone scan, and/or computed tomography (CT) if the plain x-ray is negative for fracture, especially in the presence of spondylosis, ankylosing spondylitis (AS), or diffuse interstitial skeletal hyperostosis (DISH).

Stingers and Transient Paresis

29. Every person who complains of symptoms of a "stinger" (i.e., pain and/or electrical feelings radiating down one arm following an impact) should be evaluated on an individual basis in terms of circumstances of injury, symptoms, radiographic findings, and previous history.

Associated Conditions and Injuries

The Tertiary Trauma Survey

30. Complete a comprehensive tertiary trauma survey in the patient with potential or confirmed spinal cord injury.

Traumatic Brain Injury

31. In the patient with acute spinal cord injury, particularly higher cervical injury, assess and document early and frequently any evidence of traumatic brain injury (TBI) in the form of loss of consciousness and posttraumatic amnesia. Start the assessment in the prehospital setting, if appropriate, or the emergency department.

Limb Injuries

32. Perform early stabilization of extraspinal fractures.

Chest and Abdominal Injuries

33. Screen for thoracic and intra-abdominal injury in all patients with spinal cord injury. Consider placing a nasogastric tube for abdominal decompression.

Arterial Injuries

34. In high-energy injuries, consider the possibility of an aortic injury.
35. Consider screening with CT or MR angiography for cerebrovascular injury in patients with a cervical spinal cord injury.

Penetrating Injuries

36. In the presence of penetrating injuries to the neck or trunk such as stab or gunshot wounds, perform a careful neurological examination and screen for spinal injury.
37. Remove the cervical collar while maintaining inline stabilization to attend to major neck wounds or to perform life-saving procedures after cervical injury (large vessel injury or airway obstruction), as needed.
38. Administer local wound care to stab and gunshot wounds to the spine. Provide proper antibiotic coverage; bullet fragments usually do not need removal.

Surgical Procedures

39. Perform a closed or open reduction as soon as permissible on patients with bilateral cervical facet dislocation in the setting of an incomplete spinal cord injury. If traction reduction is not preferred or possible, then open reduction should be performed.
40. Consider early surgical spinal canal decompression in the setting of a deteriorating spinal cord injury as a practice option that may improve neurologic recovery, although there is no compelling evidence that it will. Consider early spinal stabilization where indicated.

Anesthetic Concerns in Acute Spinal Cord Injury

41. Secure the airway, support respiratory status, and consider postoperative ventilatory support.
42. Maintain mean arterial pressure (MAP) and perfusion with a balance of infusion and inotropes.
43. Anticipate bradycardia and hypotension during intubation of the tetraplegic patient.

44. Avoid the use of succinylcholine after the first 48 hours post-cord injury.
45. Monitor temperature, warm intravenous (IV) fluids, and use a patient-warming device as needed.
46. Consider the use of intraoperative spinal cord monitoring in the patient with sparing of spinal cord function.

Pain and Anxiety: Analgesia and Sedation

47. Minimize the pain of allodynia. Minimize evoked pain through thoughtful patient handling.
48. Assess the patient's pain, preferably using a self-reported numeric rating scale.
 - Minimize reliance on report by family members, who may underestimate pain.
 - If using a pain rating scale based in part on the physiologic manifestations of stress associated with pain, recognize that some people with SCI and higher lesions may be unable to show changes in heart rate and blood pressure assessed by the pain score.
 - Provide adequate analgesia unless specific contraindications exist.
 - Consider short-acting sedation to allow periodic neurologic assessment.
49. Employ contemporary medical guidelines to manage pain and distress in ventilated patients with SCI.
50. Consider the use of breath-controlled analgesia in the tetraplegic patient.

Secondary Prevention

Patient Handling and Skin Protection

51. Assess areas at risk for skin breakdown frequently.
52. Place the patient on a pressure-reduction mattress or a mattress overlay, depending on the patient's condition. Use a pressure-reducing cushion when the patient is mobilized out of bed to a sitting position.
53. Provide meticulous skin care:
 - Reposition to provide pressure relief or turn at least every 2 hours while maintaining spinal precautions.

- Keep the area under the patient clean and dry and avoid temperature elevation.
 - Assess nutritional status on admission and regularly thereafter.
 - Inspect the skin under pressure garments and splints.
54. Educate the patient and family on the importance of vigilance and early intervention in maintaining skin integrity.

Prevention and Treatment of Venous Thromboembolism

55. Apply mechanical compression devices early after injury.
56. Begin low molecular weight heparin or unfractionated heparin plus intermittent pneumatic compression, in all patients once primary hemostasis is evident. Intracranial bleeding, perispinal hematoma, or hemothorax are potential contraindications to the administration of anticoagulants, but anticoagulants may be appropriate once bleeding has stabilized.
57. Consider placing a vena cava filter only in those patients with active bleeding anticipated to persist for more than 72 hours and begin anticoagulants as soon as feasible.

Respiratory Management

58. Monitor patients closely for respiratory failure in the first days following spinal cord injury.
- Obtain baseline respiratory parameters (vital capacity, FEV1) and arterial blood gases when patients are first evaluated and at intervals until stable.
 - Consider mechanical ventilation for patients with tetraplegia.
 - Admit patients with complete tetraplegia and injury level at C5 or rostral to an intensive care unit.
59. Perform a tracheotomy early in the hospitalization of patients who are likely to remain ventilator dependent or to wean slowly from mechanical ventilation over an extended period of time, unless the treating center has special expertise in the use of noninvasive ventilation.

60. Treat retained secretions due to expiratory muscle weakness with manually assisted coughing ("quad coughing"), pulmonary hygiene, mechanical insufflation-exsufflation, or similar expiratory aids in addition to suctioning.
61. Initiate a comprehensive protocol to prevent ventilator-associated pneumonia in patients with acute SCI who require mechanical ventilation for respiratory failure.

Genitourinary Tract

62. Place an indwelling urinary catheter as part of the initial patient assessment unless contraindicated. If contraindicated, use emergent suprapubic drainage instead.
63. Leave indwelling urinary catheters in place at least until the patient is hemodynamically stable and strict attention to fluid status is no longer needed.
64. Priapism is usually self-limited in acute SCI and does not require treatment. There is no evidence to support avoidance of a urethral catheter in the presence of priapism secondary to acute SCI.

Gastrointestinal Tract

65. Initiate stress ulcer prophylaxis.
66. Evaluate swallowing function prior to oral feeding in any acute SCI patient with cervical spinal cord injury, halo fixation, cervical spine surgery, prolonged intubation, tracheotomy, or concomitant TBI.

Bowel Care

67. Initiate a bowel program as recommended in the clinical practice guideline Neurogenic Bowel Management in Adults with Spinal Cord Injury.

Nutrition

68. Provide appropriate nutrition when resuscitation has been completed and there is no evidence of ongoing shock or hypoperfusion.
- Use enteral nutrition rather than parenteral nutrition.
 - Feed a standard, polymeric enteral formula initiated within 24 to 48 hours after admission, using the semirecumbent position when possible to prevent aspiration.

- Determine the caloric requirements for nutritional support in acute SCI using a 30-minute energy expenditure measurement by indirect calorimetry (metabolic cart).

Glycemic Control

69. Maintain normoglycemia in critically ill mechanically ventilated patients.

Prognosis for Neurological Recovery

70. Within the first 72 hours, use the clinical neurological assessment as described by the International Standards for Neurological Classification of SCI to determine the preliminary prognosis for neurological recovery.
71. If the clinical exam is unreliable, MRI findings or electrodiagnostic studies may be useful for determining prognosis.

Rehabilitation Intervention

72. Develop protocols that allow rehabilitation specialists to become involved early in the management of persons with SCI, immediately following injury during the acute hospitalization phase.
73. Prescribe interventions that will assist the recovery of persons with SCI, including preventive measures against possible secondary complications. Educate patients and families about the rehabilitation process and encourage their participation in discharge planning discussions.
74. Use nonpharmacologic and pharmacologic interventions for orthostatic hypotension as needed. Mobilize the patient out of bed to a seated position once there is medical and spinal stability. Develop an appropriate program for out-of-bed sitting. Limit in-bed and out-of bed semireclined sitting, as it often produces excessive skin shear and predisposes to pressure ulcer formation.

Psychosocial and Family Issues

75. Assess mental health in general and possible risk for psychosocial problems after admission and throughout acute care stay. Involve members of the health-care team as needed. Pay particular attention to the following factors:
 - Current major depression, acute stress disorder/posttraumatic stress disorder

(PTSD), or substance intoxication and withdrawal.

- Social support network (or lack thereof).
 - Cognitive functioning and learning style.
 - Personal and cultural preferences in coping style and social support.
 - Concurrent life stressors.
 - Concomitant health problems, medical conditions, medications, and history of TBI.
 - History of mental illness, including major depression, PTSD, substance abuse.
 - Use of psychiatric medications.
76. Foster effective coping strategies, health-promotion behaviors, and independence through a variety of ongoing interventions.
 - Use assistive devices such as head-controlled call bells, bed controls, prism glasses, and communication boards.
 - Acknowledge that feelings of gratitude, uncertainty, loss, and helplessness may be present simultaneously.
 - Provide medical and prognostic information matter-of-factly, yet at the same time leave room for hope.
 - Respect expressions of hope. Avoid direct confrontations of denial concerning probable implications of the injury.
 - Help the patient and family to identify effective coping strategies that have aided them in the past.
 - Develop a partnership of patient, family, and health-care team to promote involvement in the treatment plan and optimize patient outcomes.
 77. Detect suicidal ideation and requests for assisted suicide. Take treatment refusals and requests for withdrawal of treatment very seriously.
 - Acknowledge the patient's suffering.
 - Assess for and treat underlying depression, substance abuse, or other chronic condition.
 - Determine the patient's decision-making capacity.
 - Identify patient needs jointly and establish a plan of care.
 - Ensure informed consent.

- Consult the institution's ethics committee when appropriate.
- Consult legal counsel if the conflict continues or if there is any uncertainty regarding the patient's request.

Special Mechanisms of Injury

78. Screen for SCI in the patient with high-voltage electrical injury.
79. Suspect spinal cord injury in any scuba or commercial diver presenting with neurologic symptoms. Consult with and consider urgent transfer to a hyperbaric unit.

Hysterical Paralysis

80. Consider the diagnosis of hysterical paralysis in patients with marked inconsistencies in neurologic findings.
 - Repeat the neurologic exam with great care. Consider using the Spinal Injuries Center test and review base screening imaging, such as plain x-rays.
 - Consult in person or by phone with an SCI specialist before making this diagnosis.
 - Encourage the patient gently to resume normal function, minimizing disability.
 - Resort to more intensive tests, such as MRI or motor-evoked potential testing, if the patient fails to improve in 2 to 3 days.
81. Consider referral to rehabilitation professionals once confident of the hysterical paralysis diagnosis.

The Consortium for Spinal Cord Medicine

Seventeen organizations, including Paralyzed Veterans of America (Paralyzed Veterans), joined in a consortium in June 1995 to develop clinical practice guidelines in spinal cord medicine. Currently, 22 member organizations comprise the consortium. A steering committee governs consortium operation, leading the guideline development process, identifying topics, and selecting panels of experts for each topic. The steering committee is composed of one representative with clinical practice guideline experience from each consortium member organization. Paralyzed Veterans provides financial resources, administrative support, and programmatic coordination of consortium activities.

After studying the processes used to develop other guidelines, the consortium steering committee unanimously agreed on a new, modified, clinical/epidemiologic, evidence-based model derived from the Agency for Healthcare Research and Quality. The model is:

- Interdisciplinary, to reflect the multiple informational needs of the spinal cord medicine practice community;
- Responsive, with a time line of 12 to 36 months depending on the complexity of the issue to be addressed; and
- Reality-based, to make the best use of the time and energy of the busy clinicians who serve as panel members and field expert reviewers.

The consortium's approach to the development of evidence-based guidelines is both innovative and cost efficient. The process recognizes the specialized needs of the national spinal cord medicine community, encourages the participation of both payer representatives and consumers with SCI, and emphasizes the use of graded evidence available in the international scientific literature.

The Consortium for Spinal Cord Medicine is unique to the clinical practice guidelines field in that it employs highly effective management strategies based on the availability of resources in the health-care community, it is coordinated by a recognized national consumer organization with a reputation for providing effective service and advocacy for people with spinal cord injury and disease, and it includes third-party and reinsurance payer organizations at every level of the development and dissemination processes. The consortium expects to

initiate work on two or more topics per year, with evaluation and revision of previously completed guidelines as new research demands.

Guideline Development Process

The guideline development process adopted by the Consortium for Spinal Cord Medicine consists of 12 steps, leading to panel consensus and publication. After the steering committee chooses a topic, a panel of experts is selected. Panel members must have demonstrated leadership in the topic area through independent scientific investigation and publication. Following a detailed explication and specification of the topic by select steering committee and panel members, consultant methodologists review the international literature, prepare evidence tables that grade and rank the quality of research, and conduct statistical meta-analyses and other specialized studies, as needed. The panel chair then assigns specific sections of the topic to the panel members based on their area of expertise. Writing begins on each component using the references and other materials furnished by the methodology support group, with necessary additional references selected by the panel members and graded by the methodologists.

After the panel members complete their sections, the panel generates a draft document at its first full meeting. The panel incorporates new literature citations and other evidence-based information not previously available. At this point, charts, graphs, algorithms, and other visual aids, as well as a complete bibliography, are added, and the full document is sent to legal counsel for review.

After legal analysis to consider antitrust, restraint-of-trade, and health-policy matters, clinical experts from each of the consortium organizations plus other select clinical experts and consumers review the draft document. The review comments are assembled, analyzed, and entered into a database, and the document is revised to reflect the reviewers' comments. The draft document is distributed to all consortium organization steering committee members. Final technical details are negotiated among the panel chair, members of the organizations' boards, and expert panelists. If substantive changes are required, the draft receives a final legal review. The document

is then ready for editing, formatting, and preparation for publication.

The benefits of clinical practice guidelines for the spinal cord medicine practice community are numerous. Among the more significant applications and results are the following:

- Clinical practice options and care standards.
- Medical and health professional education and training.
- Building blocks for pathways and algorithms.
- Evaluation studies of guidelines use and outcomes.
- Research gap identification.
- Cost and policy studies for improved quantification.
- Primary source for consumer information and public education.
- Knowledge base for improved professional consensus building.

Methodology

Retrieval and Grading of the Scientific Evidence

BACKGROUND

Spinal cord injuries are one of the most debilitating and devastating injuries, with an estimated annual incidence of 11,000 cases per year in the United States (National SCI Statistical Center, 2006). Between 225,000 and 296,000 individuals currently live with an SCI in this country alone. Early acute management includes diagnosis, treatment, and prevention of complications, with the goals being to limit the extent of injury, manage acute consequences of the injury, and initiate measures to prevent predictable complications.

OBJECTIVE

This review is intended to provide panel members developing this guideline with the best evidence on acute injury management and to assist panel members with assessment of the strength of evidence for their recommendations. United BioSource Corporation (UBC) provided methodologic support for the development of this guideline by conducting a systematic review of the recent English-language literature on early (within 72 hours of injury) management of patients with spinal cord injuries, including diagnostic, preventive, and therapeutic interventions. Specifically, the

advantages and indications, disadvantages and contraindications, and impact on prevention of spinal cord injury complications were sought.

METHODOLOGY

UBC performed a systematic review of the literature published since 1995 that describes early acute management of spinal cord injuries in the adolescent and adult population. Procedures for this review followed the best methods used in the evolving science of systematic review research. Systematic review is a scientific technique designed to minimize bias and random error by employing a comprehensive search process and a preplanned process for study selection.

LITERATURE SEARCH

The literature search included both electronic and manual components. Medline (via PubMed) was searched back to 1995 for citations using the following Medical Subject Heading [MeSH] terms and keywords:

1. *Spinal cord injuries [MeSH] OR paraplegia/rehabilitation OR quadriplegia/rehabilitation*
2. *Acute OR early OR intensive care units [MeSH] OR trauma centers [MeSH] OR emergency medicine [MeSH] OR neuroprotective agents/therapeutic use OR emergency treatment [MeSH] OR emergency medical service [MeSH]*
3. *#1 AND #2 limits: publication date from 1995 to 2006 English, human, NOT reviews, letters, editorials*

In addition, two strategies were used to identify papers that may not have been indexed on Medline by the time of the search cutoff date. The PubMed search included a keyword search for the prior 6 months, using terms indicating spinal cord injury and early acute management, with no limits; and Current Contents was searched for the past year, using similar search terms.

The Cochrane Library and the National Guidelines Clearinghouse were searched for any recent systematic reviews of clinical guidelines on the subject that could have been sources for further references. A manual check of the reference lists of all accepted papers and of recent reviews was performed to supplement the above electronic searches. Abstracts from the electronic search were downloaded and evaluated using the literature review process described below.

STUDY SELECTION

To be eligible for inclusion in this review, studies contained none of the following exclusion criteria and each of the inclusion criteria:

Exclusion Criteria

- Abstracts, letters, comments, editorials, reviews, or surveys
- Animal and in vitro studies
- Languages other than English
- Study published before 1995
- No trauma (e.g., disease-related spinal cord lesions)
- Studies dealing with radiology protocols for cervical spine clearance

Inclusion Criteria

Studies were included if BOTH of the following criteria were met:

- Traumatic spinal cord injury or suspected spinal cord injury in patients age 13 or older
- Acute (preferably within 72 hours) or subacute (preferably within 1 week) spinal cord injury diagnosis, treatment, or management intervention studies

SEARCH YIELD

The searches yielded 1,227 abstracts. After all of the abstracts were downloaded, a level 1 screening was performed, in which abstracts were reviewed for exclusion criteria. The full article was then obtained for all accepted abstracts and for those abstracts for which a clear determination could not be made at level 1 screening. The full articles of 275 accepted studies underwent a level 2 screening, in which inclusion and exclusion criteria were applied. On completion of level 2 screening, all accepted articles were then eligible for data extraction. Any studies rejected at this level were reviewed by two researchers and listed in a reject log. This process resulted in 60 papers being accepted for data extraction, with an additional 3 papers being linked publications (additional publications for a given cohort of individuals).

DATA EXTRACTION AND DATABASE DEVELOPMENT

Data extraction forms (DEFs) were designed specifically for this project. Data extraction involves the capturing of various data elements from each study and is performed by one investigator. A second investigator establishes a consensus for all extracted data, and a third party arbitrates disagreements, as necessary. The consensus versions of the

DEF were entered into MetaHub™, UBC's relational database of clinical trials information.

After 100% of the entered data were validated against the consensus DEFs and full consistency and logic checks were performed on the database, the data were locked. After the data passed these quality control measures, they were used to generate evidence tables, which were delivered to Paralyzed Veterans for the panel's review.

A series of data elements were extracted, when possible, from each accepted study. These are available on request from Paralyzed Veterans.

EVIDENCE ANALYSIS

All studies accepted for data extraction were graded for level of evidence using the criteria from the Centre for Evidence-Based Medicine (www.cebm.net; accessed January 16, 2008) in Oxford, UK, described below. In addition, randomized clinical trials were assessed using the Jadad Quality Score Assessment. Industry sponsorship was also noted.

Levels of Evidence

The concept of levels of evidence grew out of the work of the Canadian Task Force for the Periodic Health Examination, in which recommendations for preventive health measures were tied to an assessment of the supporting evidence in the published literature. The assignment of levels of evidence in this review was based on the following guidance from the Steering Committee on Clinical Practice Guidelines for the Care and Treatment of Breast Cancer, published by the Canadian Medical Association:

- I. Evidence based on randomized controlled clinical trials (or meta-analysis of such trials) of adequate size to ensure a low risk of incorporating false-positive or false-negative results.
- II. Evidence based on randomized controlled trials that are too small to provide level I evidence. These may show either positive trends that are not statistically significant or no trends and are associated with a high risk of false-negative results.
- III. Evidence based on nonrandomized, controlled, or cohort studies; case series; case-controlled studies; or cross-sectional studies.
- IV. Evidence based on the opinion of respected authorities or of expert committees as indicated in published consensus conferences or guidelines.
- V. Evidence that expresses the opinion of those individuals who have written and reviewed this guideline, based on experience, knowledge of the relevant literature, and discussions with peers.

These five levels of evidence do not directly describe the quality or credibility of evidence. Rather, they indicate the nature of the evidence being used. In general, a randomized, controlled trial (level I) has the greatest credibility; however, the trial may have defects that diminish its value, and these should be noted. Evidence that is based on too few observations to give a statistically significant result is classified as level II. In general, level III studies carry less credibility than level I or II studies, but credibility is increased when consistent results are obtained from several level III studies carried out at different times and in different places.

Decisions must often be made in the absence of published evidence. In these situations, it is necessary to use the opinion of experts based on their knowledge and clinical experience. All such evidence is classified as "opinion" (levels IV and V). A distinction is made between the published opinion of authorities (level IV) and the professional opinion of those who have contributed to this guideline (level V). However, it should be noted that by the time level V evidence has gone through the exhaustive consensus-building process used in the preparation of this guideline, it has achieved a level of credibility that is at least equivalent to level IV evidence.

Grading the Guideline Recommendations

After the panel members drafted their sections of the guideline, each recommendation was graded according to the level of scientific evidence supporting it. The framework used by the methodology team is outlined in table 1. These ratings, like the evidence table ratings, represent the strength of the supporting evidence, not the strength of the recommendation itself. The strength of the recommendation is indicated by the language describing the rationale.

TABLE 1
Categories of the Strength of Evidence Associated with the Recommendations

Category	Description
A	The guideline recommendation is supported by one or more level I studies.
B	The guideline recommendation is supported by one or more level II studies.
C	The guideline recommendation is supported only by one or more level III, IV, or V studies.

Sources: Sackett, D.L., Rules of evidence and clinical recommendation on the use of antithrombotic agents, *Chest* 95 (2 Suppl) (1989), 2S-4S; and the U.S. Preventive Health Services Task Force, *Guide to Clinical Preventive Services*, 2nd ed. (Baltimore: Williams and Wilkins, 1996).

Category A requires that the recommendation be supported by scientific evidence from at least one properly designed and implemented randomized, controlled trial, providing statistical results that consistently support the guideline statement. Category B requires that the recommendation be supported by scientific evidence from at least one small randomized trial with uncertain results; this category also may include small randomized trials with certain results where statistical power is low. Category C recommendations are supported by either nonrandomized, controlled trials or by trials for which no controls are used.

If the literature supporting a recommendation comes from two or more levels, the number and level of the studies are reported (e.g., in the case of a recommendation that is supported by two studies, one a level III, the other a level V, the "Scientific evidence" is indicated as "III/V"). In situations in which no published literature exists, consensus of the panel members and outside expert reviewers was used to develop the recommendation and is indicated as the "Strength of Panel Opinion."

Grading of Panel Consensus

The level of agreement with the recommendation among panel members was assessed as either low, moderate, or strong. Each panel member was asked to indicate his or her level of agreement on a 5-point scale, with "1" corresponding to neutrality and "5" representing maximum agreement. Scores were aggregated across the panel members and an arithmetic mean was calculated. This mean score was then translated into low, moderate, or strong, as shown in table 2. Panel members could abstain from the voting process for a variety of reasons, such as lack of expertise associated with a particular recommendation.

TABLE 2
Levels of Panel Agreement with Recommendations

Level	Mean Agreement Score
Low	1.0 to less than 2.33
Moderate	2.33 to less than 3.67
Strong	3.67 to 5.0

REFERENCES FOR METHODOLOGY

- Cook, D.J., C.D. Mulrow, and R.B. Haynes. Systematic reviews: synthesis of best evidence for clinical decisions. *Ann Intern Med* 126 (1997): 376-80.
- Harris, R.P., M. Helfand, S.H. Woolf et al. Current methods of the U.S. Preventive Services Task Force. A Review of the Process. *Am J Prev Med* 20 (3 Suppl) (2001): 21-35.

Jadad, A.R., R.A. Moore, D. Carroll et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Controll Clin Trials* 17 (1996): 1–12.

Sacks, H.S., J. Berrier, D. Reitman et al. Meta-analyses of randomized controlled trials. *N Engl J Med* 316 (1987): 450–5.

Steering Committee on Clinical Practice Guidelines for the Care and Treatment of Breast Cancer. Introduction. *CMAJ* 158 (3 Suppl) (1998): S1–S2.

West, S., V. King, T.S. Carey et al. Systems to rate the strength of scientific evidence. Evidence Report/Technology Assessment No. 47 (prepared by Research Triangle Institute, University of North Carolina Evidence-Based Practice Center, under contract no. 290–97–0011). AHRQ pub. no. 02–E016. Rockville, MD: Agency for Healthcare Research and Quality, April 2002.

INTERNET SOURCES FOR GUIDELINE DEVELOPMENT

- Canadian Task Force on Preventive Health Care. frenchs@herlpitt.org; Michael Boninger www.ctfphc.org/ (accessed January 16, 2008).
- Centre for Evidence-Based Medicine, Oxford University. www.cebm.net/ (accessed January 16, 2008).
- New Zealand Guidelines Group. www.nzgg.org.nz/ (accessed January 16, 2008).
- Scottish Intercollegiate Guidelines Network. www.sign.ac.uk/ (accessed January 16, 2008).

TABLE 3
Levels of Evidence for Primary Research Question

	Types of Studies			
	Therapeutic Studies: Investigating the Results of Treatment	Prognostic Studies: Investigating the Effect of a Patient Characteristic on the Outcome of Disease	Diagnostic Studies: Investigating a Diagnostic Test	Economic and Decision Analyses: Developing an Economic or Decision Model
Level I	High-quality randomized controlled trial with statistically significant difference or no statistically significant difference but narrow confidence intervals Systematic review ² of level I randomized controlled trials (studies were homogeneous)	High-quality prospective study ⁴ (all patients were enrolled at the same point in their disease with ≥ 80% follow-up of enrolled patients) Systematic review ² of level I studies	Testing of previously developed diagnostic criteria in series of consecutive patients (with universally applied reference “gold” standard) Systematic review ² of level I studies	Sensible costs and alternatives; values obtained from many studies; multiway sensitivity analyses Systematic review ² of level I studies
Level II	Lesser quality randomized controlled trial (e.g., < 80% follow-up, no blinding, or improper randomization) Prospective ⁴ comparative study ⁵ Systematic review ² of level II studies or level I studies with inconsistent results	Retrospective ⁶ study Untreated controls from a randomized controlled trial Lesser quality prospective study (e.g., patients enrolled at different points in their disease or < 80% follow-up) Systematic review ² of level II studies	Development of diagnostic criteria on basis of consecutive patients (with universally applied reference “gold” standard) Systematic review ² of level II studies	Sensible costs and alternatives; values obtained from limited studies; multiway sensitivity analyses Systematic review ² of level II studies
Level III	Case-control study ⁷ Retrospective ⁶ comparative study ⁵ Systematic review ² of level III studies	Case-control study ⁷	Study of nonconsecutive patients (without consistently applied reference “gold” standard) Systematic review ² of level III studies	Analyses based on limited alternatives and costs; poor estimates Systematic review ² of level III studies
Level IV	Case series ⁸	Case series	Case-control study Poor reference standard	No sensitivity analyses
Level V	Expert opinion	Expert opinion	Expert opinion	Expert opinion

1. A complete assessment of the quality of individual studies requires critical appraisal of all aspects of the study design.

2. A combination of results from two or more prior studies.

3. Studies provided consistent results.

4. Study was started before the first patient enrolled.

5. Patients treated one way (e.g., with cemented hip arthroplasty) compared with patients treated another way (e.g., with cementless hip arthroplasty) at the same institution.

6. Study was started after the first patient enrolled.

7. Patients identified for the study on the basis of their outcome (e.g., failed total hip arthroplasty), called “cases,” are compared with those who did not have the outcome (e.g., had a successful total hip arthroplasty), called “controls.”

8. Patients treated one way with no comparison group of patients treated another way.

This chart was adapted from material published by the Centre for Evidence-Based Medicine, Oxford, UK. For more information, please see www.cebm.net.

EVIDENCE REVIEW

The studies' quality ratings and evidence tables were prepared for use by Paralyzed Veterans panel members in their guideline deliberations. Evidence tables consisted of by-study listings of extracted information. Patient, intervention, and outcome combinations were too heterogeneous to permit quantitative synthesis of outcomes data.

During the panel deliberations and preparation of the recommendations, it became clear that the

expert panel also drew extensively on a substantial literature base, providing support for their recommendations. Often, a recommendation is based on older studies of SCI patients, or on studies of more heterogeneous groups of acutely injured patients with or without SCI, studies that were believed to be generalizable to the early SCI population. UBC independently graded these studies.

Recommendations

Triage Protocols and Trauma Systems of Care

In 1981 the American Spinal Injury Association (ASIA) published its *Guidelines for Facility Categorization and Standards of Care*. These guidelines defined the components of a “system” for medical care of patients with spinal cord injury (SCI) based on current expertise. The American College of Surgeons document *Resources for Optimal Care of the Injured Patient* outlines the resources necessary for the provision of care to the multisystem-injured patient (U. S. Department of Health and Human Services Program Support Center, 2006b). In 1992, the Bureau of Health Services Resources, Division of Trauma and Emergency Medical Services, published through an expert consortium a document titled *Model Trauma Care System Plan*. This document, updated in 2006 by the U. S. Department of Health and Human Services Program Support Center, outlines the components necessary to ensure that “the right patient gets to the right facility, in the right amount of time.”

The evidence is now increasing in support of specific levels of care and expertise for patients at different stages after injury, but for each stage, it is important to carefully assess the evidence and to justify the cost of a specialized unit for that patient. The additional stress on patient and family when care is far from home and friends must also be considered.

Analysis of the etiology of spinal cord injury is important not only to guide spinal cord injury prevention efforts but also to the design of trauma triage and transport guidelines.

Prehospital Triage

1. **Develop appropriate guidelines for the evaluation and transport of patients with potential spinal cord injuries based on local resources. Identify regional trauma centers with special resources for the acute management of spinal cord injuries.**

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Regional prehospital triage protocols should be in place to direct acutely injured patients with potential spinal injury to accredited

trauma centers where trauma-trained surgeons are promptly available for initial evaluation and management. Acosta and colleagues (1998) found that the first 24 hours after trauma are the deadliest, and that primary and secondary injuries to the central nervous system are the leading cause of death, underscoring the importance of prompt evaluation by appropriate providers in an appropriate health-care setting. Khetarpal et al. (1999) found that the presence of a trauma surgeon on the trauma team reduced both resuscitation time and the time to incision for patients needing emergent operations.

Trauma Center

2. **Transfer the patient with a spinal cord injury as soon as possible to a Level I trauma center, as defined by the American College of Surgeons or by state statute. Given local triage protocols and guidelines relating to transportation times to trauma centers, consider taking the patient directly to a Level I center if possible in preference to passing through a Level II or III center first.**

(Scientific evidence—II/III/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: In addition to the need to preserve neurologic function in the possible presence of an unstable spine, the relatively high frequency of head injury associated with SCI suggests the need for early transfer to a Level I center. Early and rapid access to a trauma team that includes specialists in spine and brain injury is critical. Rapid access to imaging capability should include CT (computerized tomography) scans and MRI (magnetic resonance imaging).

Level of care, volume, and outcome. The volume of patients per center necessary for higher levels of competence and better outcomes has not been defined. Nathens et al. (2001) showed better outcomes in higher volume centers for penetrating intra-abdominal injury and multisystem blunt trauma but excluded spinal cord injury. Demetriades et al. (2005) described increased mortality in patients with certain major injury patterns cared for in Level II versus Level I trauma centers but did not show any worse outcome by level of care in a subgroup with tetraplegia in which a 24% in-hospital mortality rate was described. Their findings (and

the comments in the discussion) suggest that patients with tetraplegia alone could be assessed in a Level I or II trauma center; while those with multiple injuries, including pelvic trauma, penetrating thoracic or abdominal injury with hypotension, as well as tetraplegia, should bypass a Level II in favor of transfer directly to a Level I center. MacKenzie et al. (2006) showed minor but significant mortality differences in favor of level 1 trauma centers both for patients with a high Injury Severity Score (ISS) and for those with SCI. Demetriades et al. (2006) also showed better survival rates in patients with an ISS greater than 15 if they receive Level I care. Sampalis et al. (1997) showed reduced mortality in severely injured patients who bypassed a Level II center in favor of direct transfer to a Level I center. Their data (and, they said, those in the literature) would favor bypassing non-Level I hospitals when the injury has occurred within the urban limits. It is acknowledged that the resources required to maintain Level I care are more expensive and not universally available or close at hand. This group also showed that reduced mortality can be realized in a regionalized trauma system that includes spinal cord specialty units, although they did not specifically consider the outcome in SCI patients (Sampalis et al., 1999).

Mechanism of injury. Helling et al. (1999) noted the importance of low falls (defined as falls from less than 20 feet) as a potential cause of significant injuries to the head and spine. In their series of 159 patients whose initial condition did not trigger a response from the full trauma team, 48 had suffered head injury, 7 were tetraplegic, and 3 were paraplegic. Helling's group recommended that surgeons and emergency physicians be thorough in their evaluations and quick to transfer selected patients to trauma centers because of the potential seriousness and complexity of injuries occurring from low falls, particularly in elderly individuals.

Spinal Cord Injury Centers

3. **Consider directing spinal cord-injured patients expeditiously to a specialized spinal cord injury center that is equipped to provide comprehensive, state-of-the-art care. Discuss pre-transfer requirements with the referral center.**

(Scientific evidence—I/II/III/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: As soon as possible, and preferably within 24 hours, consult with the clinical liaison for a specialized SCI center (which may or

may not be a component of the regional Level I trauma center). Transfer the patient to specialized care when sufficiently medically stable to meet the criteria of the local specialized spinal injuries unit. Munro in the United States, Guttman in the United Kingdom, and Botterell in Canada were pioneer paraplegists who identified the specialized needs of the SCI patient. Their work led to the development of specialized centers of care in which a relatively uncommon but severe and costly condition could be managed optimally with a view to limiting complications of the injury and facilitating rehabilitation and community reintegration. During the 1940s, the Department of Veterans Affairs began establishing regional SCI units to meet the needs of veterans injured during World War II. The first regional SCI center funded by the U.S. government for nonveterans was organized in 1970; 13 more were established by 1979. In 1981, ASIA published standards for a spinal cord injury system of care, prescribing five major components of care: emergency medical services, a trauma center with SCI trauma unit, a rehabilitation facility with SCI trauma unit, a follow-up system, and a viable community integration activity.

The Cochrane review of spinal injuries centers (SICs) by Jones and Bagnall (2004) noted that the majority of complications in traumatic SCI can occur in the first 24 hours, but found insufficient evidence to support conclusions about the benefits or disadvantages of immediate referral versus late referral to SICs, suggesting the need for a well-designed prospective study of this question. Bagnall (an author of the Cochrane study) et al. also found in a systematic review that the available studies were all retrospective and observational in nature. Nonetheless, lesser levels of evidence, especially in the more recent literature, suggest that early referral and transfer to SICs may offer advantages (Bagnall et al., 2003).

Aito and colleagues (2003) noted that pressure ulcers and respiratory complications were much more common among patients who were treated in nonspecialized units or who experienced delays in transfer to a specialized unit. Aung and el Masry (1997) showed that patients admitted to an SIC within 1 week of injury suffered a lower rate of complications compared with those admitted later. Pagliacci et al. (2003) showed a greatly increased length of rehabilitation center stay in patients admitted with a pressure sore. They also showed a longer length of rehabilitation center stay in those patients originating from a less specialized early treatment center.

These studies suffer from at least a partial selection bias as shown by Amin et al. (2005) in that a delay to referral to an SCI unit can be related to the management of associated injuries. However, Amin et al. showed that a delay from referral to admission may be related to an inadequate number of appropriate beds and is associated with a longer overall time in hospital. The definition of what is an inappropriate delay to referral or to transfer to an SIC has not been established.

DeVivo et al. (1990) found statistically significant reductions in acute care and in total lengths of stay coupled with a highly significant reduction in pressure ulcers among patients admitted to the specialty spinal center within 1 day of injury compared with an otherwise comparable group of patients admitted to their spinal unit for rehabilitation only. Dallyan et al. (1998) showed that patients admitted to an SIC within 24 hours of injury had a lower rate of joint contractures. They also showed a link between the presence of a contracture and the occurrence of a pressure sore, both being associated with concomitant head injury. Yarkony et al. (1985) reviewed 181 spinal cord-injured patients admitted to the Rehabilitation Institute of Chicago following acute SCI. In their review, they analyzed the origin of the patients from either general hospitals or the acute care unit of a spinal center at Northwestern Memorial Hospital. They found that patients treated at the general hospital had a statistically significant increased incidence of contractures compared with the spinal center patients.

Heinemann et al. (1989) showed equivalent final outcomes for patients from specialized centers versus general hospital centers passing through their rehab center, but the overall time in hospital was greater for those not coming from a specialized acute unit because of the relatively longer time spent in the acute unit in the nonspecialized hospitals.

Length of stay may be shorter for patients with SCI admitted to a specialized system. Tator and colleagues (1993) found that patients treated in an acute spinal cord injury unit had a significant reduction in mortality, a significant reduction in length of stay, and a significant increase in neurological recovery (doubling of the neurological recovery scale). Amin et al. (2005) concluded that early liaison with a designated spinal injury center or unit is vitally important. Scivoletto et al. (2005) showed a better final outcome in those patients admitted to a rehab center earlier (< 30 days) than those admitted later.

Before a patient with a spinal cord injury is transported from one facility to another, the following protocol should be completed to ensure that the patient's condition is sufficiently stabilized:

- Spine immobilization is adequate and secure.
- The airway is clear and can be maintained during transfer; consider intubation prior to transfer if PaCO₂ is elevated or if ventilatory failure is likely to develop during a prolonged transfer.
- A chest tube is in place for any pneumo- or hemothorax, especially if air transport is considered.
- Supplemental oxygen is being administered and ventilation (spontaneous or assisted) is adequate.
- IV (intravenous fluid) is patent and infusing at the desired rate.
- Hemodynamic parameters have been stabilized and can be monitored during transport.
- When indicated, nasogastric tube is in situ, draining freely, and connected to low suction.
- Indwelling urinary catheter is in situ and draining freely.
- Skin is protected from injury due to excessive pressure, especially over bony prominences, such as the sacrum, that contact the support surface, and any apparatus or debris that could cause pressure sores is cleared away.
- Neurological level and completeness of injury, as determined from a motor and sensory examination according to the International Standards *Neurological Classification of SCI* (see page X), are documented immediately prior to transferring the patient.
- All imaging and other records accompany the patient.

Adapted with permission from <http://commons.bcit.ca/elearning/Managing%20SCI.zip>.

Spinal Stabilization during Emergency Transport and Early In-Hospital Immobilization Following Spinal Cord Injury

Rapid and safe transport of the spinal injury patient allows for early medical stabilization and institution of measures designed to preserve and potentially improve ultimate neurologic outcome. Interestingly, there is a lack of data from randomized controlled trials to support the practice of prehospital spinal immobilization in trauma patients. Only level III studies are available to support the use of spine immobilization for all patients with a

suspected spinal injury. Although not supported by higher levels of medical evidence, this time-tested practice is based on anatomic, mechanical, and clinical considerations in an attempt to prevent further, or new onset, spinal cord injury.

4. Immobilize the spine of all patients with a potential spinal injury from the scene of the injury to definitive care.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Management of a patient with potential spinal injury begins at the scene. It has been estimated that up to 25% of spinal cord injuries occur after the initial traumatic insult, either during transit or early in the course of management (Toscano, 1988). During the 1970s, most patients (55%) referred to regional spinal cord injury centers arrived with complete neurological lesions (Gunby, 1981); in the 1980s, more spinal cord-injured patients arrived with incomplete lesions.

Although there is no strong clinical evidence to support immobilization in spinal cord-injured patients, there is panel consensus that this should be the initial treatment. Support for spinal immobilization was presented by Toscano (1988), where 32 (26%) of 123 trauma patients experienced major neurological deterioration following injury and prior to admission to the hospital due to what was described as a lack of adequate spinal immobilization. A retrospective chart review covering a 5-year time span, performed in collaboration by the University of New Mexico in the United States and the University of Malaysia–Kuala Lumpur, looked at the efficacy of spinal immobilization and found no significant protective effects from spine immobilization (Hauswald, Ong *et al.*, 1998). However, Hadley's review (2002) notes the limitations of this report but also comments on the dearth of sufficient evidence to support practice standards. Hadley presents options for neck protection during extrication and transportation, recommends early removal of protective devices once definitive management is established and notes the need for ongoing research in this area.

A number of reports have been published over the past few decades criticizing current methods of immobilization due to sporadic instances of adverse occurrences in a small percentage of cases (Domeier, 1999). What is needed is a clear and uniform protocol for immobilization and transport of patients with both suspected and proven spinal column and cord injury to minimize further neurologic demise and reduce costs to the health-care system.

5. Emergency medical service (EMS) providers should use the following five clinical criteria to determine the potential risk of cervical spinal injury in a trauma patient:

- Altered mental status.
- Evidence of intoxication.
- Suspected extremity fracture or distracting injury.
- Focal neurological deficit.
- Spinal pain or tenderness.

(Scientific evidence–I/II/IV; Grade of recommendation–A; Strength of panel opinion–5)

Rationale: Domeier *et al.* (1999) found in a multicenter prospective study of 6,500 trauma patients that the application of any one of these clinical criteria was predictive in the majority of patients with cervical spinal injuries of the need for immobilization. The predictive value of their criteria held true for patients with high- or low-risk mechanisms of injury. The investigators suggested that clinical criteria, rather than mechanism of injury, be evaluated as the standard for deciding whether to use spine immobilization.

When determining if EMS providers are indeed able to apply clinical criteria to evaluate the stability of the cervical spine, L. H. Brown *et al.* (1998) found that EMS personnel and hospital-based physicians have a moderate to substantial agreement when determining the stability of the cervical spine. Their assessments correlated in 79% of the cases ($n = 451/573$). For 78 patients (14%), the EMS clinical assessment indicated spine immobilization, while the physician assessment in the trauma bay did not. For only 44 patients (8%), the physician's clinical assessment indicated spine immobilization, while the EMS assessment did not. For individual components, the correlation coefficient ranged from 0.35 to 0.81. For the decision to immobilize, it was 0.48. The EMS clinical assessments were generally more in favor of immobilization than the physician's clinical assessments later in the emergency room, erring on the side of safety during patients' pre-hospital care. Similar evidence does not exist for thoracolumbar injuries.

Patients with a significant head injury are also at risk of spinal cord injury and should be carefully evaluated for the presence of a cervical cord lesion. Holly and colleagues (2002) assessed the risk of cervical spine trauma associated with moderate and severe head injury. They noted that 5% of a series of 447 patients with moderate or severe head injury had sustained a significant cervical

spinal injury; 58% of those had a cervical spinal cord injury, often in the upper cervical spine. They also noted a much greater chance of these injuries in patients with a Glasgow Coma Scale (GCS) of 8 or less, and in those with a vehicular mechanism of injury. In a similar population of 41,142 patients with traumatic brain injury, Piatt (2006) reported that the prevalence of cervical injury was 8%. Provider vigilance is therefore required to evaluate for spinal injury and acute SCI in patients with traumatic brain injury. Prasad et al. (1999) also noted the predilection for injury at the C1 level in those cervical-injured patients with a GCS of less than 5.

6. EMS providers should use the combination of rigid cervical collar immobilization with supportive blocks on a backboard with straps or similar device to secure the entire spine of patients with potential spinal injury.

(Scientific evidence—I/II; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: As many as 20% of spinal column injuries involve multiple noncontiguous vertebral levels. Therefore, the entire spinal column is potentially at risk after trauma. As a consequence, complete spine immobilization and cross-body strapping is recommended in the transport of patients from the injury scene.

In 1985, Cline et al. found that strapping a patient to a standard short board was more effective than using a cervical collar alone in attempting to immobilize the cervical spine. They noted no significant differences among the different rigid collars that were also tested in this study. Perry et al. (1999) confirmed these data, observing that the efficacy of cervical spine immobilization was limited unless the motion of the head and the trunk were also effectively controlled. It appears that a combination of rigid cervical collar immobilization with supportive blocks on a rigid backboard with straps to secure the entire body is the most effective method to limit spinal motion following trauma (De Lorenzo, 1996; American Association of Neurological Surgeons and the Congress of Neurological Surgeons [AANS and CNS], 2002). Preexisting spine deformities must be accommodated when immobilizing the patient (see also recommendation 12).

7. In the emergency department, transfer the patient with a potential spinal injury as soon as possible off the backboard onto a firm padded surface while maintaining spinal alignment.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: A rigid backboard should be used for as short a period of time as possible for initial inpatient evaluation and stabilization (Vickery, 2001). Prompt removal from the backboard, after transport to an emergency department and initial spine stabilization, is required to reduce pressure ulcer formation (AANS and CNS, 2002). For patients with a confirmed spinal cord injury, transfer the patient off the backboard onto a firm padded surface, ideally within 2 hours, continuing precautions to protect the spinal column and skin. Those who have extended transport to the emergency department or who are delayed in transfer to the intensive care unit are at increased risk of skin breakdown (Consortium for Spinal Cord Medicine, 2000).

8. In cases of confirmed spinal or spinal cord injury, maintain spine immobilization until definitive treatment.

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Hard cervical collars may provide appropriate immobilization for some injuries; however, considerable concern has arisen regarding their prolonged use in patients with severe head and multiple injuries. Even after a few days of use in this setting, there have been reports of occipital and submental decubiti, raised intracranial pressure, and increased risk of aspiration (Davies et al., 1996; Mobbs et al., 2002).

Historically, once the radiological diagnosis of a bony injury has been established, initial treatment has been directed toward spine immobilization, and if appropriate, spinal realignment. The first modern immobilization device—Crutchfield cranial tongs—was introduced in 1933 and was in subsequent clinical use for more than four decades. Gardner-Wells tongs became available in the 1970s and largely supplanted previous devices because of their ease of placement and more versatile clinical applicability. Halo immobilization became popular in the 1960s, although it was not widely used as an acute immobilization device until several decades later and now is the primary method of acute stabilization. The titanium halo ring offers the advantage of allowing CT and MRI scans to be performed and may be readily converted to an orthosis to provide definitive treatment of the spine injury (Wilberger, 2000). Although there are a variety of studies of the biomechanical and kinematic stability of the halo orthosis for long-term stabilization, there are no comparable studies in the acute setting. Effective thoracolumbar immobilization can be achieved by maintaining the patient on a firm padded surface and using appropriate techniques for transfers or repositioning.

9. At the extremes of age, or in the presence of a preexisting spine deformity, provide patient care in the position of greatest comfort while maintaining immobilization.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Although there is no evidence to determine the optimum method and position of support for the head of the spine-injured person, the premorbid spine contour (which varies with age and certain spinal conditions such as ankylosis) and the mechanism of injury may determine the best position. Certainly, allowing too much extension (e.g., on a flat surface without head support) for the older person with a kyphotic cervicothoracic spine, or too much flexion (e.g., in the small child with a large head nursed on a flat surface) can increase the risk of neurologic deterioration after cervical fracture. The safest position until imaging is completed is often that of greatest comfort for the injured person (e.g., a support of comfortable height such as a folded sheepskin under the head and neck of an older patient with a thoracic kyphosis).

10. Employ an adequate number of personnel during patient transfers for diagnostic studies and for repositioning to maintain the alignment of a potentially unstable spine and avoid shearing of the skin.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Imaging and diagnostic studies during the emergency and early acute period following injury often require transferring the patient from a stretcher to an imaging table. It is essential to maintain alignment of the spine and also prevent shearing injury to the skin during movement. Rigid slides or other transfer devices or sheets can assist in these efforts. No evidence was found, but ideally four people are involved in completing the transfer: one to stabilize the head and direct the transfer, two people to assist with the trunk and limbs, and one person to manage the device itself (British Trauma Society, 2003).

11. Logroll the patient with a potentially unstable spine as a unit when repositioning, turning, or preparing for transfers.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Maintaining alignment of the spine is paramount to prevent further injury and discomfort. Removal from the long backboard, placement

of sheets or devices prior to the transfer, and preparations for procedures may require moving the patient whose spine has not been cleared. Four or five people may be required to logroll while maintaining alignment: one to stabilize the head and coordinate the transfer, two people to assist with the trunk, one person to move the limbs, and one person to place or remove the device itself (British Trauma Society, 2003).

12. Consider a specialized bed for the patient with an unstable spine when prolonged immobilization is anticipated.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: For injured patients who have an unstable spine or who may require extended immobilization, the use of specialized beds and other protective surfaces can decrease the risk of complications and morbidity, including skin breakdown (British Trauma Society, 2003). Although there are no studies involving spinal cord-injured patients, evidence showing a reduction in complications due to immobility in critically ill and trauma patients suggests that there may be some benefit in patient outcomes and a decrease in costs associated with morbidity.

13. Initiate measures to prevent skin breakdown if prolonged time on a backboard is anticipated.

(Scientific evidence–II/IV; Grade of recommendation–B; Strength of panel opinion–5)

Rationale: Provide pressure relief over bony prominences every 30 minutes if it is anticipated that the patient will be maintained on a backboard for longer than 2 hours. Those who are repositioned with pressure relief during the initial 2 hours following injury are less likely to experience skin breakdown. Length of immobilization on a long rigid backboard is significantly associated with the development of pressure ulcers (Consortium for Spinal Cord Medicine, 2000; Linares et al., 1987; Mawson et al., 1988). Some degree of pressure ulcer formation occurs in 30%–50% of patients with new SCI during the first month post-injury, and the sacrum is the most common location for these ulcers. Pressure ulcers can delay the remobilization of patients during rehabilitation and force modification of the sitting program. Myocutaneous flap surgery for pressure ulcers is sometimes needed before a patient can begin sitting in a wheelchair, and a severe ulcer can delay full participation in rehabilitation for 3 months or longer.

14. Perform a baseline skin assessment on removal of the backboard.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Visually inspect the entire dorsal surface of the body, with particular attention to bony prominences, when logrolling the patient to remove the backboard. Document the baseline skin assessment, and institute preventive measures for any at-risk areas, such as reddened skin, bony prominences of the scapulae, sacrum, and the heels (see also, *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals*, Consortium for Spinal Cord Medicine, 2000).

‘ABCs’ and Resuscitation

Spinal cord injury often does not exist in isolation. Other traumatic and medical conditions of the patient must be considered when selecting management strategies. As with all trauma patients, the acute management of a patient with SCI requires rapid restoration of the airway, breathing, and circulation.

15. Provide an airway and ventilatory support in patients with high tetraplegia early in the clinical course.

(Scientific evidence–IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Patients with motor-complete injury at a level rostral to C5 will almost invariably require ventilatory support. Intense monitoring for respiratory failure is warranted in all patients with cervical spine injury (Velmahos et al., 2003). A variety of management options may be employed for support of ventilation and endotracheal intubation (Consortium for Spinal Cord Medicine, 2005). Endotracheal intubation can be particularly difficult in the patient with SCI, especially if the lesion is in the cervical spine. In addition, intubation frequently needs to be accomplished before the presence or location of an injury can be confirmed. As a result, everyone who needs urgent endotracheal intubation following trauma should be treated as if he or she has a cervical spine injury. The goal of intubation is to secure the airway with as little movement of the cervical spine as possible. The standard urgent or emergent intubating technique for someone with a presumed or known cervical spine injury is a rapid sequence induction with cricoid pressure and manual inline stabilization. For further information, refer to www.asahq.org/Newsletters/2005/11-05/2003

TraumaAlgorithm.html and www.asahq.org/Newsletters/2005/11-05/wilson11_05.htm/ (accessed January 18, 2008).

The choice of induction agent and neuromuscular blocking agent requires some consideration, however. Although propofol and thiopental are commonly used as induction agents in hemodynamically stable patients, both may exacerbate hypotension resulting from hemorrhage, neurogenic shock, and sepsis. Ketamine and etomidate remain viable alternatives in these settings. Ketamine, which may actually cause hypertension, is controversial in patients with concomitant head injury due to longstanding concern that it may elevate intracranial pressure (Wyte, 1972). Etomidate provides stable hemodynamics during induction, but there is concern about the safety of its use in critically ill patients (D. Annane, 2005). Etomidate inhibits adrenal steroid synthesis (R.L. Wagner et al, 1984); P Cohan et al., 2005) and has been associated with hypotension and need for vasopressors, but the clinical significance of this is controversial. Retrospective studies showing worse outcomes in patients who receive etomidate may be biased in that sicker patients are more likely to receive etomidate at induction. Until this issue is investigated further, a reasonable approach might be to use etomidate for induction in the multiple-trauma patient or when tenuous hemodynamics are present. Should refractory shock develop, exogenous steroids should be considered. With respect to neuromuscular blocking agents, succinylcholine remains the agent of choice for rapid sequence intubation in SCI patients within the first 48 hours of injury. Following this time window, a nondepolarizing neuromuscular blocking agent should be used instead.

If a difficult intubation is anticipated, an awake fiberoptic intubation is an appropriate alternative, and other methods may also be necessary (see www.asahq.org/Newsletters/2005/11-05/2003 TraumaAlgorithm.html and www.asahq.org/Newsletters/2005/11-05/wilson11_05.html/; accessed January 18, 2008.) Furthermore, an awake fiberoptic intubation may be the preferred method of securing the airway in a cooperative patient who does not have impending respiratory failure, as it is possible to accomplish without any movement of the cervical spine, and the patient can undergo a brief neurological exam immediately following completion of the procedure.

If airway and mechanical ventilatory support are not required, consider evaluation of baseline pulmonary function on admission with measurement of tidal volume, vital capacity, and negative inspiratory force so that follow-up assessments

can be compared with the individual's baseline for early diagnosis of acute respiratory failure.

16. Prevent and treat hypotension.

(Scientific evidence–II/IV; Grade of recommendation–B; Strength of panel opinion–4)

Rationale: Early appropriate fluid resuscitation is necessary for all patients with SCI to maintain tissue perfusion, but care must be taken to avoid fluid overload. The first treatment priority for hypotension is fluid resuscitation. The goal is to maintain optimal tissue perfusion and to resolve shock. The appropriate resuscitation end point and optimal mean arterial blood pressure for maintenance of spinal cord perfusion are not known. Uncontrolled studies that used fluids and vasopressors to achieve a mean arterial pressure of 85 mmHg for a minimum of 7 days in patients with acute SCI have reported favorable outcomes (Levi et al., 1993; Vale et al., 1997).

Hypotension may exacerbate central nervous system injury. Avoiding hypotension in brain-injured patients is paramount in early treatment because diminished cerebral perfusion pressure may contribute to secondary neuronal injury (R. M. Chesnut, 1993). Although separate clinical data do not exist for patients with spinal cord injuries, hypotension should be recognized, the cause of the hypotension sought, and fluid resuscitation initiated with the goal of treating hypotension (systolic blood pressure < 90). Further study is needed to define ideal mean arterial pressure (MAP) and the potential role for elevation of MAP with fluids or pharmacologic treatment (Vale et al., 1997).

17. Determine initial base deficit or lactate level to assess severity of shock and need for ongoing fluid resuscitation.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–4)

Rationale: Standard hemodynamic parameters (blood pressure, pulse) do not adequately quantify the degree of shock and physiologic derangement in trauma patients, particularly in those with SCI. Initial base deficit or lactate level can be used to determine the severity of shock and the need for ongoing fluid resuscitation. The optimal algorithms for fluid resuscitation, blood product replacement, and the use of inotropes and/or vasopressors have not been determined; further research in this area is needed, particularly for patients with spinal cord injury. Resuscitation algorithms aimed at achieving supranormal oxygen delivery or preventing splanchnic ischemic reperfusion injury have not been determined to be efficacious in a general

trauma ICU population as assessed with various types of monitoring and several clinically relevant outcome measures. For detailed discussion of this issue, see www.east.org/tpg.asp/ (accessed January 18, 2008; Tisherman et al., 2004).

18. Exclude other injuries before assigning the cause of hypotension to neurogenic shock.

(Scientific evidence–III/IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Acute SCI may be associated with hemodynamic instability. Neurogenic shock (reduced blood pressure from neurologic causes) is common in patients with acute tetraplegia or high-level paraplegia (T1–T4), but before assuming that the cause of hypotension is from the cord injury, other causes of hypotension should be investigated. The clinician managing traumatic SCI should be attentive to all potential causes of hemodynamic instability, including hemorrhage, pneumothorax, myocardial injury, pericardial tamponade, sepsis related to abdominal injury, and other traumatic and medical etiologies. Physical examination and subjective patient reports are problematic in the insensate spinal cord injured patient, and chest/abdomen/pelvis CT or other imaging modalities should be performed to exclude other possible causes of hypotension.

Other potential causes of hypotension, such as adrenal insufficiency, should also be considered. In a study of 80 patients with moderate or severe traumatic brain injury, approximately 50% of patients had transient adrenal insufficiency. Younger age, greater injury severity, early ischemic insults, and the use of etomidate and metabolic suppressive agents were associated with adrenal insufficiency (Cohan et al., 2005). Evaluation for adrenal insufficiency with a cosyntropin (ACTH) stimulation test may be warranted (Garcia-Zozaya, 2006).

19. Recognize and treat neurogenic shock.

(Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: A patient with acute SCI may present with or develop neurogenic shock (Gondim et al., 2004; Krassioukov and Claydon, 2006). This occurs secondary to sympathetic denervation, resulting in arteriolar dilation and pooling of blood in the venous compartment, and interruption of cardiac sympathetic innervation (T1–T4) with unopposed vagal activity promotes bradycardia and reduced myocardial contractility. Neurogenic shock is suggested by decreased blood pressure and systemic vascular resistance with a variable heart rate response (Bilello et al., 2003; Bravo et al., 2004;

Gondim et al., 2004). Experimental data indicate that hypotension and shock are particularly deleterious to the injured spinal cord, contributing to cord hypoperfusion and perpetuating secondary cord injury (AANS and CNS, 2002).

Higher levels of SCI correlate with more severe hypotension. The loss of vasoconstrictor tone in the peripheral arterioles is associated with pooling of blood in the peripheral vasculature. In the setting of neurogenic shock, it is essential to first ensure that intravascular volume is restored, then vasopressors (dopamine, norepinephrine, phenylephrine) may be used to treat hypotension (Stevens et al., 2003).

20. Monitor and treat symptomatic bradycardia.

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Cardiovascular intervention may be required for patients with cervical SCI and tetraplegia (Bilello et al., 2003). Acute cervical SCI may also result in bradydysrhythmias, which may lead to hypotension and asystole (Abd and Braun, 1989; Dixit, 1995; Lehmann et al., 1987). Such symptoms are more common in the first 2 weeks after injury. Bradycardia may also occur and is often associated with a noxious stimulus such as endotracheal suctioning. Cardiovascular interventions, such as the use of vasopressors, atropine, aminophylline, or pacemakers, are more commonly required in high cervical injury patients (Bilello et al., 2003; Franga et al., 2006; Pasnoori and Lessar, 2004; Ruiz-Arango et al., 2006). Vasopressors should be chosen so as to minimize exacerbation of bradycardia. An ideal agent should have both alpha- and beta-adrenergic actions, such as dopamine, norepinephrine, or epinephrine, to counter the loss of sympathetic tone and provide chronotropic support to the heart.

21. Monitor and regulate temperature.

(Scientific evidence—I/II/III; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: The autonomic nervous system is disrupted in cervical and high thoracic SCI (above T6), resulting in altered thermoregulation due to loss of vasomotor control and sympathetically mediated vasodilation. Poikilothermia results, which is a state in which the body assumes the temperature of the surrounding ambient environment. People with SCI above T6 may experience hypothermia as well as reduced ability to dissipate body heat. Although no studies conducted during the acute phase of SCI were found, research indicates that impaired thermoregulation may persist for years after an injury (Nicotra et al., 2006

Webborn et al., 2005; Price and Campbell, 2003). Therefore, monitoring temperature is essential during the acute management phase.

Neuroprotection

Pharmacologic Neuroprotection in Patients with Spinal Cord Injury

For decades, physicians have tried to improve final neurologic outcomes in patients following spinal cord injury. Acute traumatic SCI involves both primary and secondary mechanisms of injury. The primary mechanism involves the initial mechanical injury due to local deformation and energy transformation that occurs within the spinal cord at the moment of injury. This insult is irreversible. Secondary mechanisms of injury occur shortly after the initial traumatic event and lead to tissue destruction during the first few hours after injury. These secondary mechanisms include processes such as ischemia, axonal degeneration, and inflammation, which are potentially preventable and/or reversible. Therefore, the concept of targeting secondary mechanisms of injury is a key element in the development of neuroprotective therapies to improve neurologic recovery after acute SCI.

22. No clinical evidence exists to definitively recommend the use of any neuroprotective pharmacologic agent, including steroids, in the treatment of acute spinal cord injury to improve functional recovery.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Over the past several years, a number of human clinical trials have evaluated the efficacy of potential neuroprotective therapies following traumatic spinal cord injury; however, none of these studies has conclusively shown a benefit in preserving or improving spinal cord function. Large-scale, multicenter clinical trials have investigated the neuroprotective impacts of methylprednisolone (MP), the neuroganglioside GM-1, gacyclidine (aspartate receptor antagonist), tirilazad (free radical scavenger), and naloxone. Unfortunately, undisputed efficacy of these agents has never been demonstrated, and with the risk of possibly severe side effects, their use cannot be recommended following SCI. At this time, there is also no evidence for the clinical use of hypothermia. The reader is advised to check for new therapies through the resources of the U.S. National Library of Medicine (PubMed) and for studies that are recruiting through the U.S.

National Institutes of Health (<http://clinicaltrials.gov/ct2/results?term=acute+spinal+cord>; accessed January 2008).

Methylprednisolone. MP has been investigated in three large-scale, multicenter clinical trials collectively referred to as NASCIS (National Acute Spinal Cord Injury Study; Bracken et al., 1984, 1990, 1997). The first NASCIS trial compared 2 doses of MP after traumatic SCI (Bracken et al., 1984); the second clinical trial compared the effects of a much higher dose of MP with those of naloxone and a placebo (Bracken et al., 1990); and the third clinical trial evaluated the timing of initiation and duration of MP treatment following injury (Bracken et al., 1997). Although improvements in ASIA motor and sensory scores were reported in subjects who received MP in both the NASCIS II and NASCIS III trials, neither study addressed some of the potential confounding variables, which limits the extent to which the results of the studies can be generalized to all individuals with acute spinal cord injuries (Hurlbert, 2000, 2006). For example, the NASCIS II trial did not include details about other interventions such as radiology, surgical manipulations, or the extent of rehabilitative therapies, which may have contributed to improvements or recovery. Furthermore, subsequent post hoc analysis failed to demonstrate improvement in primary outcome measures (motor scores, pinprick scores, and light-touch scores), meaning that improved recovery with MP may represent random events, thus weakening the overall study findings (Coleman et al., 2000; Hurlbert, 2000, 2006; Short et al., 2000).

It is also important to note that MP can have significant side effects. Both the NASCIS II and III studies documented serious complications associated with MP administration, such as higher infection and sepsis rates, respiratory complications, and gastrointestinal hemorrhage. MP therapy should not be initiated more than 8 hours after the SCI and has not been shown to be effective in SCI caused by penetrating (gunshot) trauma (Bracken et al., 1997).

GM-1. The ganglioside GM-1 is another neuroprotective agent that has been investigated for use after acute SCI in humans. GM-1 is a lipid that is abundant in mammalian central nervous system membranes. Its proposed mechanism of action lies in its ability to prevent apoptosis and to induce neuronal sprouting in the setting of spinal cord injury. Although basic science investigational data demonstrated enhanced neuronal plasticity, regeneration, and a neuroprotective effect following its administration, preclinical experimental data were very limited regarding

the efficacy of GM-1 in the setting of spinal cord injury. Various animal studies have reported improvements in neurologic recovery after traumatic SCI following the administration of GM-1 (Sygen, Fidia Pharmaceutical Corporation, Washington, DC). Although a single-center clinical study suggested a benefit to its use when administered on a daily basis for 1 month following SCI, the findings from a large-scale, multicenter clinical trial with 760 subjects did not demonstrate a benefit in ASIA-impairment grade (principal end point) for treated patients compared with individuals who received a placebo (AANS and CNS, 2002); and a Cochrane review failed to find any benefit (Chinnock and Roberts, 2005).

Gacyclidine. A large SCI trial in France investigated the efficacy of gacyclidine, an N-methyl-D-aspartate receptor antagonist that demonstrated a neuroprotective effect in animal models of SCI. A total of 200 patients were randomized to either treatment with gacyclidine or a placebo within 3 hours of injury. All subjects underwent, if necessary, surgical decompression and stabilization. Subjects were examined via blinded assessments over a 1-year period. Results reported thus far have demonstrated no statistically significant improved neurologic outcomes in those patients who received gacyclidine. However, subjects with an incomplete cervical spinal cord injury appeared to show neurologic improvement with its use (Steeves et al., 2004).

Tirilazad and naloxone. In the NASCIS II and III trials, naloxone and tirilazad were investigated for their neuroprotective properties in clinical application after SCI. Although a very small, nonfunctional, motor-only improvement of one grade was seen in the group treated with tirilazad within 8 hours from the time of SCI, no statistically significant improvements were found, similar to naloxone (Bracken et al., 1997).

Other promising pharmaceutical agents currently undergoing investigation include a tetracycline derivative, minocycline (phase II investigation in Calgary, Canada), and erythropoietin, the hormone that regulates erythropoiesis. In general, encouraging basic science animal studies have not always shown similarly positive outcomes in human clinical studies. Additionally, it is important to realize that certain therapeutic interventions may potentially worsen the natural course of SCI in research subjects; that the vast majority of therapeutic agents, although promising in animal models, will never demonstrate efficacy in human trials; and that interactions between agents may be of concern. Fortunately, the clinical studies that have evaluated the protective effects of MP, GM-1, and gacyclidine have proven that large-

scale, prospective clinical trials are feasible and that the encouraging preclinical results for minocycline and erythropoietin can be evaluated using such models to determine their efficacy in the treatment of spinal cord injury.

23. If it has been started, stop administration of methylprednisolone as soon as possible in neurologically normal patients and in those whose prior neurologic symptoms have resolved to reduce deleterious side effects.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Administration of MP to patients without spinal cord injury is not without risk and certainly has no benefit. Complications of high-dose steroid use include increased infection rates, sepsis, wound-healing complications, pulmonary embolism, peptic ulcer disease, hyperglycemia, and lipid profile changes. It can cause severe reactions in patients with type I diabetes and steroid-induced myopathy. Although only shown in patients with preexisting vascular conditions, avascular necrosis of the femoral and/or humeral heads can be caused by high-dose steroid use, but was not demonstrated in an MRI study of humeral and femoral heads in steroid-treated spinal cord patients (Wing et al., 1998).

In conclusion, MP therapy should never be started in neurologically normal patients or in any patient beyond 8 hours from the time of SCI. MP can cause significant side effects in the injured patient with no compelling evidence that it improves neurologic outcome.

Diagnostic Assessments for Definitive Care and Surgical Decision Making

Clinical Neurologic Assessment for SCI

24. Perform a baseline neurological assessment on any patient with suspected spinal injury or spinal cord injury to document the presence of SCI. If neurologic deficits are consistent with spinal cord injury, determine a neurological level and the completeness of injury. Perform serial examinations as indicated to detect neurological deterioration or improvement.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: In a patient who is awake and cooperative, the clinical neurological examination of strength and sensation is the recommended method for diagnosing and classifying SCI. The instrument most widely used in SCI centers for standardized assessment and classification is the International Standards for Neurological Classification of SCI, also commonly referred to as the ASIA standards (see figure 1). This classification requires a clinical examination with manual muscle testing of 10 key muscles bilaterally, a sensation examination for light touch and sharp/dull discrimination in 28 dermatomes bilaterally, and a rectal examination for sensation and voluntary contraction. Early classifications derived from this examination have prognostic value in determining the likelihood for neurological recovery (see the section “Prognosis for Neurological Recovery”), and they have sufficient interrater reliability to be used in clinical trials of strategies to improve neurological outcomes following SCI.

The neurological level is classified as the lowest level with normal function, provided all rostral levels are normal. The completeness of injury is classified using the ASIA Impairment Scale grade, commonly referred to as the ASIA score, which replaced a prior grading system known as Frankel grades. ASIA A indicates a complete injury, with complete loss of sensory and motor function below the cord lesion. ASIA B indicates complete loss of motor but some preservation of sensation below the injury level, as determined by the presence of sensation in the S4–S5 dermatome or on rectal exam. ASIA C and ASIA D refer to injuries that are sensory and motor incomplete; in ASIA C, the majority of the muscles below the neurological level are less than 3/5 on manual muscle testing, and in ASIA D, at least 50% of the muscles below the neurological level are 3/5 or greater. ASIA E indicates that the neurological examination is normal (see figure 1).

Because of the progressive evolution of neurological deficits, neurological examinations should be repeated after transport and following such procedures as the application of traction or reduction maneuvers to monitor for deterioration or improvement. The frequency of repeat neurological examinations must be individualized, based on the clinical status of the patient and on the protocols of the institution, but in the first 3 days the exam will be performed at least once daily.

Patient Name _____
 Examiner Name _____ Date/Time of Exam _____



STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY



MOTOR

KEY MUSCLES (scoring on reverse side)

	R	L	
C5	<input type="checkbox"/>	<input type="checkbox"/>	Elbow flexors
C6	<input type="checkbox"/>	<input type="checkbox"/>	Wrist extensors
C7	<input type="checkbox"/>	<input type="checkbox"/>	Elbow extensors
C8	<input type="checkbox"/>	<input type="checkbox"/>	Finger flexors (distal phalanx of middle finger)
T1	<input type="checkbox"/>	<input type="checkbox"/>	Finger abductors (little finger)
UPPER LIMB TOTAL (MAXIMUM) <input type="checkbox"/> + <input type="checkbox"/> = <input type="text"/> (25) (25) (50)			

Comments:

L2	<input type="checkbox"/>	Hip flexors
L3	<input type="checkbox"/>	Knee extensors
L4	<input type="checkbox"/>	Ankle dorsiflexors
L5	<input type="checkbox"/>	Long toe extensors
S1	<input type="checkbox"/>	Ankle plantar flexors

Voluntary anal contraction (Yes/No) ☐ ☐

LOWER LIMB TOTAL (MAXIMUM) ☐ + ☐ = (25) (25) (50)

SENSORY

KEY SENSORY POINTS

0 = absent
1 = impaired
2 = normal
NT = not testable

	LIGHT TOUCH		PIN PRICK	
	R	L	R	L
C2				
C3				
C4				
C5				
C6				
C7				
C8				
T1				
T2				
T3				
T4				
T5				
T6				
T7				
T8				
T9				
T10				
T11				
T12				
L1				
L2				
L3				
L4				
L5				
S1				
S2				
S3				
S4-5				

TOTALS: ☐ (56) ☐ (56) = (max: 112)

Any anal sensation (Yes/No) ☐ ☐

PIN PRICK SCORE (max: 112)

LIGHT TOUCH SCORE (max: 112)

NEUROLOGICAL LEVEL: The most caudal segment with normal function

SENSORY: ☐ R ☐ L

MOTOR: ☐ R ☐ L

COMPLETE OR INCOMPLETE? ☐

Incomplete = Any sensory or motor function in S4-S5

ASIA IMPAIRMENT SCALE: ☐

ZONE OF PARTIAL PRESERVATION: Caudal extent of partially innervated segments

SENSORY: ☐ R ☐ L

MOTOR: ☐ R ☐ L

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REV 03/06

ASIA IMPAIRMENT SCALE

- ☐ **A = Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.
- ☐ **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.
- ☐ **C = Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
- ☐ **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
- ☐ **E = Normal:** Motor and sensory function are normal.

CLINICAL SYNDROMES (OPTIONAL)

- ☐ Central Cord
- ☐ Brown-Sequard
- ☐ Anterior Cord
- ☐ Conus Medullaris
- ☐ Cauda Equina

Radiographic Evaluation of Patients Following Spinal Cord Injury

Initial imaging protocols are frequently dependent on the presenting circumstances of the trauma patient, the experience of the treating physicians and institutions, and available resources. The goal of spinal trauma imaging is to detect all injuries using the least amount of resources with the least potential harm to the patient. Accordingly, cost-effective diagnostic imaging modalities that allow early detection of spinal injury with a high negative predictive value would ensure safe

and effective early care of the spinal trauma patient. This section applies to patients who have clear signs or symptoms of spinal cord injury.

25. Image the entire spine in a patient with SCI.

(Scientific evidence—I/II/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Imaging should include a multi-slice CT protocol of the entire spine to delineate the known injury and to exclude noncontiguous injuries. If CT is not available, perform three views of all the regions of the spine with conventional antero-posterior and lateral plain radiographs.

It is especially important to image the lower cervical spine and cervicothoracic junction. In the face of a known spinal injury, from 10% to 40% of patients may show a noncontiguous injury depending on the imaging technique used, putting the entire spinal column potentially at risk (Qaiyum et al., 2001; Vaccaro, 1992).

The most prevalent initial radiographic assessment of the symptomatic or obtunded patient has been the 3-view cervical spine series. When the entire cervical spine from the occiput to T1 has been visualized, the negative predictive value of a normal study has been reported to range from 93% to 98% in several level I studies (Ajani et al., 1998; Berne et al., 1999), and from 85% to 100% in level II and III studies (Borock et al., 1991). Although the negative predictive value of the 3-view cervical spine radiographs is quite high, the sensitivity is much less impressive. Sensitivity rates between 63% and 83% have been reported by these same studies (Ajani et al., 1998; Berne et al., 1999). In the best-case clinical scenario, approximately 98% of patients with a normal 3-view cervical spine series will have a truly normal cervical spine. The much lower sensitivity rates suggest that the radiograph series will be normal in approximately 15% of patients who have a true cervical spine injury. In total, this could lead to the “normal” interpretation of abnormal radiographs in 1 per 100 patients with a true injury (McCulloch et al., 2005).

The most common cause of missed cervical spine injury seems to be failure to adequately visualize the region of injury. This occurs most commonly at the extremes of the cervical spine (i.e., the occiput to C2 and the C7–T1 levels; Davis et al., 1993). In a retrospective study, Vaccaro et al. (1992) described 372 spinal injury cases admitted to a regional spine center where 3% of spinal injury cases were initially missed on plain films at an outside institution, and 25% of these were associated with progressive neurologic deficit. Davis et al. (1993) described 32,117 acute trauma patients in which cervical spine injuries were missed 34 times despite symptoms. Twenty-three of these 34 symptomatic patients either did not have radiographs or had inadequate radiographs that did not include the region of injury. Eight patients had adequate radiographs that were simply misread by the treating physician. Only one patient had a missed injury that was undetectable on technically adequate films, even after retrospective review.

Until the beginning of the 1990s, plain radiographs were the initial imaging tools used to assess bony injury and malalignment after SCI. It was reported that in approximately 50% to 70%

of all cases, plain radiographs were sufficient to identify all existing spinal injuries. However, several authors found that conventional radiographs alone could miss 23% to 57% of fractures of the cervical spine. This significant percentage may result in catastrophic neurologic worsening in the peritrauma period if an unstable spinal lesion is missed and the patient is inadequately immobilized.

To increase the sensitivity of the radiographic assessment of the cervical spine, regardless of neurologic status, many authors have described the added utility of CT in the acute trauma setting. Greater injury detection sensitivity has been reported with CT, especially in spinal regions not well visualized on plain films, typically the cranio-cervical and cervicothoracic junctions, or areas identified as suspicious on plain cervical spine radiographs (Berne et al., 1999). The negative predictive value and sensitivity of CT to detect unstable spinal fractures have been reported to be 100% (level II and III data); however, these studies have used CT itself as the gold standard. This represents a false endpoint for the true variable of a clinically relevant spinal injury.

Spiral or helical CT has become a popular screening imaging tool throughout North America in the setting of spinal trauma and those thought to be at risk for spinal injury. High-risk patients are described as those with multiple injuries, those with abnormal mental status, or those whose mechanism of trauma suggests spinal injury. In a recent prospective study, McCulloch et al. (2005) described 407 patients who underwent evaluation for cervical spine trauma by conventional radiographs with or without the addition of helical CT imaging. Despite rating the 3-view cervical radiographs as acceptable for clearance of the cervical spine in 194 cases (48%), 12 of 25 (48%) spinal injuries were missed when compared with CT imaging. In one patient, an odontoid fracture was missed by helical CT, although it was identified on conventional plain radiographs since because the fracture line was parallel to the axial CT image.

When a cervical fracture is identified, imaging of the entire spine must be completed. In a study of the National Trauma Databank, more than 190,000 patients who had sustained injuries in a motor vehicle crash were identified (Winslow et al., 2006). Of these, 8% had cervical spine fractures, 5% had thoracic spine fractures, 6% had lumbar spine fractures, and 10% had either thoracic or lumbar fractures. Of patients with a cervical spine fracture, 13% also had a thoracic or lumbar fracture, whereas among patients without cervical spine fracture, only 7% had a thoracolumbar fracture. The odds ratio for a thoracolumbar fracture

in the presence of a cervical spine fracture was 2.02 ($p < .0001$). These data confirm a strong association between cervical spine fractures and thoracolumbar fractures after blunt vehicular trauma and support the practice of imaging the complete spine when a cervical fracture is identified.

26. Perform an MRI of the known or suspected area(s) of spinal cord injury.

(Scientific evidence—I/IV/V; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: MRI provides excellent soft-tissue and spinal cord imaging and is useful in identifying the presence of specific soft-tissue injuries often seen in the setting of neurologic injury. Often, MRI will give clues as to the causes of neurologic injury, such as spinal cord contusion or stretch, which cannot be illustrated by plain radiography or CT.

Following blunt injury, approximately 54% of neurologically incomplete patients and 36% of complete patients will have a disk injury detected by MRI (Rizzolo et al., 1994). MRI has also been found to have a high sensitivity and negative predictive value in detecting injury to spinal ligaments and soft tissues. Benzel et al. (1996) used an ultra-low-field MRI magnet to evaluate patients whose physical examination or plain radiography was equivocal for spinal injury. The investigators found that 16% of patients had both disk and ligamentous disruption and that 20% had isolated ligamentous abnormalities.

T2 fat-suppressed MRI has been shown to identify almost double the number (41%) of visualized contiguous and noncontiguous vertebral fractures compared to with plain films (10%–15%; Gupta and el Masri, 1989; Qaiyum et al., 2001). This implies that current imaging strategies relying solely on plain radiography to assess for noncontiguous injuries may be inadequate. It appears that if MRI is indicated in the setting of spinal injury, a rapid MRI assessment (sagittal T2 image) of the whole spine is practical to avoid missing an occult spinal injury at a distant level.

Lastly, MRI has been shown to be able to identify a high incidence (26%) of significant ligamentous, disk, or bony injury in obtunded and ventilated patients with negative plain films in the ICU setting. These findings may be used to further direct CT evaluation. Even when a good quality helical or multislice cervical CT is normal, MRI has been found to detect spinal abnormalities, including ligamentous injuries, in 10% of high-risk patients. Vaccaro et al. (1999) found that routine MRI screening of both conscious and unconscious patients was only cost effective in

the setting of a neurological deficit. Although more than half the study patients were eventually excluded, MRI findings changed the management of patients in about 25% of cases with a neurologic deficit. Currently, the American College of Radiology recommends MRI as the imaging modality of choice in an unconscious patient with a normal CT and radiographic evaluation following trauma to assess for cervical spine instability, as opposed to traction-lateral radiography as practiced in some institutions (Anderson et al., 2000).

Premorbid Spinal Conditions and the Extremes of Age: The Mobile and the Stiff Spine

Premorbid spine conditions may influence the pattern of injury resulting from a mechanical force to the spine. Variations in spine anatomy as well as the mechanical properties associated with the extremes of age and with disease states can affect the nature of any associated injuries.

27. In patients with SCI, be aware that bony imaging of the spinal column may be negative (i.e., “SCIWORA,” or SCI without radiological abnormality).

(Scientific evidence—II; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: Not all SCI is associated with a spinal fracture or dislocation. In some patients, SCI may result from forced extreme range of spinal movement without mechanical failure of the spinal column. R. L. Brown et al. (2001) reported that spinal cord injury without radiographic abnormality comprised 38% of a series of 103 consecutive patients admitted to a Level I pediatric trauma center. Brown’s study suggested that a high index of suspicion for SCIWORA is essential when evaluating adolescents with sports-related neck trauma or victims of child abuse. Be particularly alert for SCI in the child who may be suffering physical abuse.

28. In a patient with a stiff spine and midline tenderness, suspect a fracture. Consider MRI, bone scan, and/or CT if the plain x-ray is negative for fracture, especially in the presence of spondylosis, ankylosing spondylitis (AS), or diffuse interstitial skeletal hyperostosis (DISH).

(Scientific evidence—II/III/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: People in these categories are at increased risk of sustaining a spine fracture leading to cord damage. These fractures may be undisplaced initially and later displace, leading to delayed

onset cord damage. Spivak et al. (1994) demonstrated that relatively low-velocity falls may produce a central cord syndrome in people with an abnormal spine that is stiff and/or stenotic (i.e., in cervical spondylosis). The risk for a person with AS suffering a spine fracture is several times greater (Cooper et al., 1994; Feldtkeller et al., 2006; Hitchon et al., 2002; Mitra et al., 2000) and/or developing spinal cord injury is 11.4 times greater than in the general population. Esophageal injury may be seen in this group in association with cervical injury, while (less commonly seen) the potential for aortic injury exists in association with thoracic or lumbar fractures in the ankylosed spine (Lifshutz et al., 2005). Be aware also of the potential for SCI in the patient with other spine-weakening pathology (e.g., rheumatoid arthritis).

Stingers and Transient Paresis

29. **Every person who complains of symptoms of a “stinger” (i.e., pain and/or electrical feelings radiating down one arm following an impact) should be evaluated on an individual basis in terms of circumstances of injury, symptoms, radiographic findings, and previous history.**

(Scientific evidence—II/V; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Unilateral upper extremity stingers are the most frequent cervical spine-related injury in football. The annual incidence has been reported to be 8% (Castro et al., 1997). Symptoms affect a single upper extremity and are often transient, lasting from a few minutes to a couple of days. Symptoms are due to a peripheral nervous system injury and may involve unilateral paresthesias, pain, and weakness. If the patient is an athlete, return to play criteria are controversial—there are no generally accepted guidelines on return to play for athletes after stinger injuries.

Transient tetraparesis may imply cord injury and should lead to evaluation of the patient by a spine specialist with appropriate imaging (Allen and Kang, 2002).

Associated Conditions and Injuries

Spinal cord injury can result from high- or low-energy mechanisms. The most common high-energy mechanisms are motor vehicle crashes and falls from a height, both of which may cause other major injuries that significantly influence mortality and morbidity. Rehabilitation can be severely hampered by a concomitant closed head injury, for example, and a careful screen for associated injuries early in the patient's hospital course is

essential. Most patients with a spinal cord injury will have other injuries requiring treatment.

The Tertiary Trauma Survey

The tertiary trauma survey is defined by the American College of Surgeons as a patient evaluation that identifies and catalogs all injuries after the initial resuscitation and operative intervention. The tertiary trauma survey begins with a comprehensive review of the medical record with emphasis on the mechanism of injury and pertinent comorbid factors such as age. It includes repetition of the primary and secondary surveys with a complete head-to-toe evaluation. The survey is completed with an examination of all laboratory data and a review of all radiographic studies with an attending radiologist. Any new physical findings will require further studies to rule out missed injuries. A standardized worksheet that catalogs all injuries is completed and becomes part of the patient's hospital record. At that point, a comprehensive care plan is developed.

The timing of the tertiary trauma survey differs from institution to institution, but typically it occurs within 24 hours after admission and is repeated when the patient is awake, responsive, and able to communicate any complaints. The evidence suggests that further longitudinal prospective studies are needed to identify optimal timing of the survey.

30. **Complete a comprehensive tertiary trauma survey in the patient with potential or confirmed spinal cord injury.**

(Scientific evidence—II/III/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: Not all multisystem injuries are detected in patients at the time of admission. Spinal and spinal cord injuries may initially be missed because of a combination of factors (Poonnoose et al., 2002), yet the incidence of concomitant extraspinal fractures with acute SCI is high. In their review of a large consecutive sample of people with new SCI from the National SCI Database from 1986 to 1995, Wang et al. (2001) found that 28% of the patients had extraspinal fractures and that 37% of these had more than one fracture site. The most common region for fractures was the chest, followed by lower extremity, upper extremity, head, and pelvis. Chen and DeVivo's (2005) review of more than 18,000 patients with SCI from 24 model systems from 1973 to 1999, with prospectively gathered data from the first 24 hours postinjury, showed about 28% had an extraspinal fracture. This figure rose to 50% for motorcyclists and 62%

for pedestrians struck by automobiles and fell to 3% for divers (Chen and DeVivo, 2005). Although sternal fractures are most often associated with unstable thoracic fractures (the “fourth spinal column”), Vioreanu et al. (2005) reported that spinal injury at the lower thoracic, upper lumbar, and cervical levels may also be associated with sternal injuries.

Enderson and colleagues (1990) found an injury using the tertiary trauma survey that had been missed in almost 10% of a series of unselected admitted trauma patients and noted that impaired sensorium or emergency surgery before completion of a secondary survey were possible reasons for these injuries being missed. The lack of sensation in tetraplegia and traumatic brain injury with their resulting lack of symptoms increases the likelihood of a missed fracture. Buduhan and McRitchie (2000) noted that patients with missed injuries tend to be more severely injured with initial neurologic compromise.

Traumatic Brain Injury

- 31. In the patient with acute spinal cord injury, particularly higher cervical injury, assess and document early and frequently any evidence of traumatic brain injury (TBI) in the form of loss of consciousness and posttraumatic amnesia. Start the assessment in the prehospital setting, if appropriate, or the emergency department.**

(Scientific evidence—II/III/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: In addition to the GCS, the patient should be administered the Galveston Orientation and Amnesia Test (GOAT) or an equivalent, as a sensitive marker of TBI indicating the need for later neuropsychological testing. The tests may be performed serially. Early identification and quantification of cognitive deficits may warrant later detailed neuropsychological assessment and facilitate rehabilitation planning. (Contemporary guidelines to the management of TBI may be found at www.braintrauma.org/; accessed January 11, 2008.)

Patients with acute spinal injury have a high incidence of TBI. However, Davidoff et al. (1985a, 1985b) noted a variable rate of documentation of loss of consciousness (LOC) and posttraumatic amnesia (PTA) in emergency and subsequently in rehabilitation settings. Roth et al. (1989) noted varying periods of PTA in 35 of 81 spinal cord injured patients. Davidoff et al. (1988b) found that 49% of 82 spinal cord injured patients had TBI documented by LOC or PTA of any duration; the incidence was 3.7 times higher in those injured

in road traffic accidents than all other etiologies combined. Early identification and quantification of cognitive deficits may warrant later detailed neuropsychological assessment and facilitate rehabilitation planning. Davidoff et al. (1988a) determined that the GOAT is a reliable instrument to evaluate patients at high risk for TBI. In a series of 34 patients admitted 1.7 ± 0.9 days after injury, the GOAT was administered serially at the same time each day for 3 to 5 days or until a score of 90 was achieved. It provided a sensitive method of assessing for PTA, acknowledging that although abnormalities in GOAT scoring may relate to hypoxia or drugs, they still suggest the possibility of TBI.

In a study of 468 patients with cervical injury drawn from a polytrauma population, Prasad et al. (1999) showed that only 60% of those with spinal injuries at C4, C5, C6, and C7 had a GCS of 13 or more. The investigators also noted a high association of C1–C2 spinal injury with major facial trauma. Iida et al. (1999) showed that 35% of 188 patients with cervical spine and/or spinal cord injuries had moderate or severe TBI (GCS < 13). Brain damage, skull base fractures, and severe intracranial hematomata were more frequently associated with upper than lower cervical injury.

Limb Injuries

- 32. Perform early stabilization of extraspinal fractures.**

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Extraspinal fractures occurring concurrently with a spinal cord injury are not pathological fractures in weakened bone; they are fractures sustained in a usually healthy person. The goal should be for the highest standard of fixation, although undisplaced low-energy fractures may be managed nonoperatively, taking great care to protect insensate skin by avoiding encircling casts and nonremovable splints. Perform this surgery as early as possible to facilitate early rehabilitation and concomitantly with any required spinal stabilization if the patient is medically stable.

Rogers and Shokes (2005) and Garland et al. (1985, 1986) have shown a lower rate of complications with early surgical treatment of femoral and tibial shaft fractures sustained in the same incident producing the cord injury. It is especially important to maintain the range of motion of involved joints to provide the cord injured person as much independence as possible.

Chest and Abdominal Injuries

33. Screen for thoracic and intra-abdominal injury in all patients with spinal cord injury. Consider placing a nasogastric tube for abdominal decompression.

(Scientific evidence—I/II/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: A careful evaluation for associated thoracic and abdominal injuries should be performed in all patients with acute SCI. The frequency of associated abdominal or thoracic injuries is variable in the literature.

Published reports have documented that only 2.6% to 4.2% of blunt trauma victims with cervical cord injury also sustained intra-abdominal injuries (Albuquerque et al., 1992; Soderstrom et al., 1983). A higher rate of associated abdominal injuries has been reported in patients with thoracolumbar spine injuries. Rabinovici et al. (1999) found significant intra-abdominal injuries in 10% of 258 patients with lumbar fractures associated with blunt trauma, injuries that were more likely in those patients with multilevel vertebral fractures. Those patients with lumbar fractures and intra-abdominal injuries also had a higher incidence of thoracic, pelvic, and traumatic brain injuries, and the incidence of long bone, extra-lumbar spine, and facial fractures was similar.

The physical examination is not reliable in patients with acute SCI because of possible insensitive abdominal and thoracic exams. Therefore, other diagnostic modalities are required. In patients with cervical SCI and hypotension, first perform an abdominal ultrasound (Focused Assessment with Sonography for Trauma, or FAST) examination on the patient (Scalea et al., 1999). FAST is used as a diagnostic tool in the evaluation of blunt abdominal trauma, particularly in hemodynamically unstable patients to look for the presence of hemoperitoneum (Farahmand et al., 2005). Alternatively, if ultrasound and FAST are not available, consider diagnostic peritoneal lavage.

In the presence of a high-risk mechanism of injury or of other major injuries (e.g., lower rib fracture, pelvic ring disruption, intrathoracic injury, femoral fracture, or dislocation) or in patients with a positive FAST examination, perform abdomen/pelvis CT. The use of FAST examination as a screening tool for blunt abdominal injury in the hemodynamically stable trauma patient results in underdiagnosis of intra-abdominal injury. The FAST examination does not readily identify intraparenchymal or retroperitoneal injuries, and a CT scan of the abdomen/pelvis is necessary to reduce the incidence of missed injuries (M. A. Brown et

al., 2001). In a study of 722 patients with blunt abdominal trauma undergoing FAST, 52 (7%) had abdominal injury, but 15 of 52 (29%) had no hemoperitoneum on their admission CT scan, and all had FAST interpreted as negative. Four patients with splenic injury required laparotomy, and 11 patients with splenic or hepatic injury were managed nonoperatively. Clinical risk factors significantly associated with abdominal injury without hemoperitoneum included abrasion, contusion, pain, or tenderness in the lower chest or upper abdomen; pulmonary contusion; lower rib fractures; hemo- or pneumothorax; hematuria; pelvic fracture; and thoracolumbar spine fracture. Up to 29% of abdominal injuries may be missed if blunt abdominal trauma is evaluated with admission FAST as the sole diagnostic tool and thoracolumbar spine fracture is a significant risk factor (Chiu et al., 1997).

Ballard and colleagues (1999) prospectively examined an algorithm whereby select patients who were considered high risk for occult injuries would undergo a CT scan of the abdomen/pelvis when the FAST exam was negative. Entrance criteria included adult patients with blunt trauma and a spine fracture (with or without cord injury) or a pelvic fracture. Of 32 patients with spine injuries \pm SCI, only one had a false-negative FAST exam. In contrast, of 70 patients with pelvic fractures, 13 had false-negative FAST exams, 9 patients required nonoperative management for solid organ injuries, and 4 patients required surgery for intra-abdominal injuries.

Similarly, Miller and colleagues (2003) documented that the use of FAST examination as a screening tool for blunt abdominal injury in the hemodynamically stable trauma patient results in underdiagnosis of intra-abdominal injury. Of 372 patients with blunt abdominal injury, 22 false-negative FAST exams were identified by CT scan imaging as the confirmatory test for abdominal injury. Six patients with false-negative FAST exams required laparotomy for intra-abdominal injuries, and 16 patients required admission for nonoperative management of intra-abdominal injuries.

Arterial Injuries

34. In high-energy injuries, consider the possibility of an aortic injury.

(Scientific evidence—II/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: Injury of the aorta in association with spine fractures may be produced by high-energy trauma (Murakami et al., 1998). Sturm et al. (1990) concluded that patients with one or more fractures of T1 to T8 have a statistically significant

increase in the incidence of thoracic aortic rupture. Aortic disruption ranging from an intimal tear to a full-thickness laceration may also be seen with blunt abdominal injury and a distractive mechanism of injury, such as may result from seat belts (Inaba et al., 2001). Urgent repair may be needed (Chui et al., 1999). Rarely, pseudoaneurysms may present later (Lifshutz et al., 2005).

35. Consider screening with CT or MR angiography for cerebrovascular injury in patients with a cervical spinal cord injury.

(Scientific evidence—I/III/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Blunt cerebrovascular injuries (BCVI), including carotid and vertebral injuries, are now diagnosed in approximately 1% of all blunt trauma patients (Cothren and Moore, 2005). Comprehensive screening of patients has resulted in the early diagnosis of BCVI during the asymptomatic phase, thus allowing treatment that could prevent neurologic sequelae. The optimal diagnostic method of routine screening for BCVI in patients with a cervical SCI remains controversial.

In a prospective 2-year study, patients (n = 216) were screened for BCVI with 4-vessel cerebral angiography using the following criteria: all patients with cervical spine fractures, LeFort II or III facial fractures, Horner's syndrome, skull base fractures involving the foramen lacerum, neck soft-tissue injury, or neurologic abnormalities unexplained by intracranial injuries. The overall screening yield was 29%, with 24 patients with carotid injuries and 43 patients with vertebral artery injuries. This aggressive screening protocol of patients with blunt head and neck trauma identified an incidence of BCVI in 1% of all blunt admissions. Early identification, which led to early treatment, significantly reduced stroke rates (Miller et al., 2002).

Cervical spine fracture patterns predictive of subsequently identified BCVI and accepted risk factors for BCVI include a high-energy transfer mechanism with cervical spine fracture patterns, including any fractures of C1–C3, subluxation, and fractures involving the transverse foramen (Cothren et al., 2003). BCVI screening with 4-vessel cerebrovascular angiography (in the “silent period” and preferably within hours or at least by the next day) is cost effective because of a significant reduction in the stroke rate with antithrombotic therapy (Cothren and Moore, 2005; Cothren et al., 2003).

Torina et al. (2005) examined the frequency of traumatic vertebral artery thrombosis in 632 blunt

trauma patients with cervical spine fractures with or without an associated SCI. Vertebral artery thrombosis was present on admission MR imaging/MR angiography (MRA) in 83 of 632 patients (13%), and 59% of these (49 of 83) had associated SCI. Vertebral artery thrombosis was more common in motor-complete patients (ASIA A and B, 20%) than in motor-incomplete (ASIA C and D, 10%) or in neurologically intact (ASIA E, 11%) cervical spine-injured patients (p = .019). The investigators concluded that the absence of neurologic symptoms in a patient with cervical spine fracture does not preclude vertebral artery thrombosis and recommended that MRA be considered in the diagnostic evaluation of these patients (Torina et al., 2005), as did Friedman et al. (1995). Similarly, Taneichi et al. (2005) reported vertebral artery occlusion with MRA in 17% of patients with cervical SCI, and 90% of these were unilateral and asymptomatic. CT angiography with newer technology also demonstrates high accuracy (99%) for diagnosis of BCVI (Eastman et al., 2006).

Penetrating Injuries

36. In the presence of penetrating injuries to the neck or trunk, such as stab or gunshot wounds, perform a careful neurological examination and screen for spinal injury.

(Scientific evidence—I; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Penetrating injuries, such as stab or gunshot wounds, may cause much local soft-tissue damage without bony damage or instability. In a prospective multicenter review of the Model Systems experience, Waters et al. (1995) noted that stab wounds accounted for 3% of their U.S. patient series. The investigators reported that 63% of 32 patients were initially noted to have motor-incomplete lesions, while 4 of 7 who initially appeared to be motor complete recovered some motor function. Peacock et al. (1977) reported a series of 450 stab wounds of the spinal cord admitted to a single South African center over 13 years, comprising 25% of their cord injured patients. Although almost 30% were to the cervical cord (16% complete lesions), the thoracic cord was most commonly affected (24% complete). Surgery was rarely used in this series for removal of retained foreign bodies. Early mobilization is usually possible.

37. Remove the cervical collar while maintaining inline stabilization to attend to major neck wounds or to perform life-saving procedures after cervical injury (large vessel injury or airway obstruction), as needed.

(Scientific evidence–IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: The risk for cervical spine instability is exceedingly low in neurologically intact, awake gunshot wound victims in the emergency department (Medzon et al., 2005).

38. Administer local wound care to stab and gunshot wounds to the spine. Provide proper antibiotic coverage; bullet fragments usually do not need removal.

(Scientific evidence–II/IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Penetrating trauma to the spine needs surgical intervention only if gross contamination is suspected but not primarily to remove missile fragments. Treat any spinal instability resulting from gunshot injuries to the spine in the usual manner appropriate to the fracture. In cases of severe contamination (transcolonic injury to the spine), proper surgical irrigation and debridement is advised, although the literature is unclear as to whether or not infection rates can be reduced. Steroids are not recommended for penetrating wound victims (Heary et al., 1996).

In a 10-year review of 2,450 patients with gunshot wounds to the head or neck, Klein et al. (2005) found that 10% had spine and/or spinal cord injury and that this was frequently unsuspected at the initial evaluation. Kihitir et al. (1991) evaluated 21 patients with transperitoneal gunshot wounds of the thoracic or lumbar spine; 11 of those patients (52%) were paraplegic on admission and 10 (48%) had a fixed partial neurologic deficit. This study also supports a high index of suspicion for spinal cord injury with deficit in torso gunshot wounds. Mirovsky et al. (2005) noted that neurologic deficit is possible even without violation of the spinal canal. Cornwell et al. (2001) reviewed 1,000 patients with torso gunshot wounds, of whom 141 had spinal column and/or cord injuries; protective immobilization was not felt to be necessary in transporting these patients, but 73 patients had complete neurologic deficit on admission; 10 patients died early from other traumatic injuries; and only 58 patients survived with vertebral column injury and/or incomplete neurologic deficit. In a 2003 study by Connell et al. of patients with penetrating trauma and spinal injury, all had either obvious

clinical evidence of an SCI or were in traumatic cardiac arrest, and all had spinal immobilization. The investigators suggested that fully conscious patients (GCS = 15) with isolated penetrating trauma and no neurological deficit do not require spinal immobilization. Kihitir et al. (1991) suggested a conservative surgical approach to the transperitoneal component of the injury; Simpson et al. (1989) noted no advantage of surgical over nonsurgical treatment and suggested the optimal treatment protocol was yet to be established. Quigley and Place (2006) reported an increased complication rate after surgical treatment of gunshot wounds over nonsurgical treatment and also noted that the optimal protocol has yet to be determined. Heary et al. (1996) noted that conservative treatment with antibiotics and tetanus prophylaxis is all that is needed after most penetrating wounds to the thoracic and lumbar spines unless there is progressive neurologic deficit or a persistent cerebrospinal fluid leak.

Surgical Procedures

Surgical intervention is commonly used to manage patients with an acute cervical spinal cord injury. This often involves reducing or realigning the spinal elements, decompressing compromised neural tissue, and stabilizing the spine. Basic science and animal experimental evidence suggests that early decompression of a compressed and injured spine may result in improved neurological recovery; however, the timing of surgical intervention has been a subject of much debate as there are no well-designed and well-executed level I studies that have determined if early (< 12 hours) versus late decompression is beneficial to spinal cord recovery.

39. Perform a closed or open reduction as soon as permissible on patients with bilateral cervical facet dislocation in the setting of an incomplete spinal cord injury. If traction reduction is not preferred or possible, open reduction should be performed.

(Scientific evidence–II/III/IV; Grade of recommendation–B; Strength of panel opinion–4.5)

Rationale: A recent review by Fehlings and Perrin (2005) highlighted a limited number of level II and III studies reporting on early surgical decompression or traction for cervical spinal cord injury. They recommended urgent decompression for the patient with locked facets. This procedure is not entirely without risk. Tator et al. (1999) reported on the attempted closed reduction of 585 cases of cervical facet dislocation, and although improvements were noted overall, an 8% rate of

neurologic deterioration occurred with early reduction. Based primarily on level II studies, it appears that patients with bilaterally locked facets and an incomplete neurologic injury benefit from an urgent reduction, preferably within 8 hours from the time of injury (Ng et al., 1999; Papadopoulos et al., 2002; Tator et al., 1999). Early research findings from MRI-guided reduction suggest that reduction can be safely achieved despite significant disk disruption (Darsaut et al., 2006). The risks and benefits of early reduction should be discussed with the patient and/or family prior to performing spinal reduction.

40. Consider early surgical spinal canal decompression in the setting of a deteriorating spinal cord injury as a practice option that may improve neurologic recovery, although there is no compelling evidence that it will. Consider early spinal stabilization where indicated.

(Scientific evidence—II/III/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: In the review cited above, Fehlings and Perrin (2005) recommended surgical intervention for neurologic deterioration. Tator et al. (1999) recently reviewed current practice patterns on the use and timing of spinal surgery after SCI in 36 North American centers. No neurologic recovery outcomes data were provided. Of 585 patients with SCI, 54% had an MRI, with 66% of these demonstrating spinal cord compression or deformation. Decompression with traction was attempted in 47%, but the technique proved effective in only 43% of patients. Surgery for decompression was subsequently performed in 68% of all patients at the following time intervals:

< 24 hours:	24%
25–48 hours:	16%
48–96 hours:	19%
> 5 days:	42%

Thus, although decompressive surgery is being carried out in significant numbers of patients, suggesting a perception that it may benefit neurologic recovery, the timing of the procedure reflects data from Marshall et al. (1987), who strongly advocated waiting 5 days before any spinal surgical intervention because of the risk of neurologic deterioration. It is, however, common clinical practice to urgently perform spinal canal decompression if the patient shows signs of ongoing neurologic deterioration.

The majority of clinical studies published to date are noncontrolled case series comparing outcomes to historical controls or arbitrary times to intervention (Papadopoulos et al., 2002). The study by Wiberg and Hauge (1988) is a common

example. These authors followed 30 patients (including an unknown number with cauda equina injuries) who all experienced neurologic improvement—at least one Frankel grade—when decompressed from 24 hours to 1 week, while only 60% improved if more than 1 week had elapsed. At the opposite end of the spectrum is the prospective, randomized study by Vaccaro et al. (1997), who found no statistically significant difference in motor score recovery in 34 patients operated on less than 72 hours from injury versus 28 patients undergoing surgery more than 5 days from injury. In this study, however, more than 20% of patients in both groups underwent posterior fusion alone.

These two studies illustrate the extreme difficulty in comparing and contrasting the large number of clinical studies that have been undertaken. First, there were significant differences in the timing of the surgeries. Second, in many cases it cannot be discerned whether the surgery was performed primarily for decompression or stabilization. Third, various systems were used to determine initial neurologic deficit and subsequent neurologic recovery. Fourth, cord injuries were frequently intermixed with cauda equina injuries. Fifth, radiographic confirmation of the degree and extent of decompression was variable. Finally, varying lengths of follow-up were provided.

In 2004 a large and much criticized meta-analysis was conducted involving 1,687 patients extracted from 108 published clinical studies from 1966 to 2000 (La Rosa et al., 2004). While finding a highly statistically significant improvement in neurologic recovery ($p < .001$) with early surgery (< 24 hours), the authors found their analysis invalid because of extreme inhomogeneities in the studies. Thus, only the findings for patients with an incomplete spinal cord injury were reliable. This meta-analysis suggested that early decompression (< 24 hours) in this group resulted in improved neurologic outcomes compared with delayed decompression (> 24 hours) or conservative management. The authors concluded that early decompression should only be considered as a practice option, which is concordant with Fehlings and Perrin's (2005) conclusion.

Since NASCIS established an 8-hour window for the putative benefits of methylprednisolone for nonpenetrating injuries (Bracken et al., 1990), several studies have attempted to match this window for spinal canal decompression. Patients with a bilateral facet dislocation or other reasons for acute spinal cord injury as confirmed by MRI were shown to derive neurological improvement from early reduction and spinal stabilization compared to control groups. Improvement in neurologic outcomes was reported in an investigation by Papadopoulos

et al. (2002) in which surgical decompression less than 10 hours from SCI resulted in clinically improved outcomes. A large number of studies suggesting that early decompression may enhance neurologic recovery in select groups of patients after SCI unfortunately lacks study randomization or appropriate controls. Therefore, these studies are generally considered only level III evidence (Fehlings and Perrin, 2005).

Multiple studies have shown that if early surgery for spinal canal decompression and/or stabilization is undertaken, it is not associated with an increased incidence of complications (Duh et al., 1994; Rosenfeld et al., 1998), including neurologic deterioration, and may have the benefits of decreasing ventilator and ICU days and time to patient mobilization. These shorter ICU stays translate into lower mean hospital costs (Rosenfeld et al., 1998). Campagnolo et al. (1997) found a significantly shorter length of stay in patients undergoing spinal stabilization less than 24 hours after injury ($p = .01$), with no increase in medical complications such as pneumonia, deep venous thrombosis, decubitus, or gastrointestinal hemorrhage. In the Papadopoulos et al. (2002) study, the protocol patients spent on average 14 fewer days in the hospital and 29 fewer total days in the hospital.

In conclusion, it remains to be determined if decompressive surgery—or reduction—within the NASCIS MP window within 8 hours confers any neurologic benefit. Although the Surgical Timing of Acute Spinal Cord Injury Study to determine if there is a clinical benefit after early surgical decompression is currently ongoing, the 1999 pilot study showed that it may be technically difficult to achieve a surgical decompression within the proposed timeframe. Delays in referral, transport, and in obtaining appropriate imaging studies, along with patients' hemodynamic stability issues, appear to be the most important obstacles preventing safe and early surgical intervention (Ng et al., 1999).

Anesthetic Concerns in Acute Spinal Cord Injury

Many medical and physiological issues are important to the safe conduct of a general anesthetic during the early surgical management of the spinal cord-injured patient, and some of these are discussed elsewhere in this guideline. This section emphasizes the most crucial procedures to be followed.

41. Secure the airway, support respiratory status, and consider postoperative ventilatory support.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Virtually all surgery for acute spinal cord injury requires general anesthesia with an endotracheal tube. If a patient arrives in the operating room without an endotracheal tube in place, the role of the anesthesiologist is to secure the airway safely without exacerbating the SCI. Intubation requires special care to ensure that the unstable spine remains aligned. Having the patient awake during intubation is often needed when there is a known or suspected cervical spine injury. The technique used for intubation will be dependent on the amount of time elapsed since injury, the NPO (nothing by mouth) status of the patient, the anticipated difficulty of the airway, the presence or absence of a cervical spine injury, and the level of comfort of the anesthesiologist with various methods. Succinylcholine remains a safe muscle relaxant for use in the first 48 hours following injury.

Intraoperative events, such as blood loss requiring transfusion and generous intravenous fluids, as well as prone positioning resulting in facial and airway edema, may further compromise a patient's respiratory status. Lung compliance may be reduced. Transfusion-related acute lung injury may develop. Surgical intervention may exacerbate an evolving neurological deficit with increased diaphragm weakness. Residual anesthetic agents and opioids may diminish already compromised airway reflexes, including cough and gag. For these reasons, the anesthesiologist should have a low threshold for transporting the patient to the intensive care unit with the endotracheal tube in place, even for patients who had reasonable respiratory status prior to the surgical procedure. Patients with lesions of C5 and above are particularly at risk for respiratory failure and will likely need postoperative mechanical ventilation.

42. Maintain MAP and perfusion with a balance of infusion and inotropes.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—4)

Rationale: As a result of autonomic instability, injuries at or above T6 are especially associated with hypotension (Raw et al., 2003). Although replacement of any lost fluid is necessary, this hypotension responds poorly to IV fluid resuscitation, which may lead to pulmonary edema. Maintenance of perfusion pressure according to current guidelines could potentially be cord-sparing. The

choice of vasopressor depends on the level of the SCI and the hemodynamics of the patient. A high thoracic or cervical spine injury characterized by hypotension and bradycardia will necessitate a drug with both chronotropic and inotropic effects, as well as vasoconstrictor properties. Dopamine and norepinephrine are both reasonable agents. For low thoracic lesions where hypotension is mainly the result of peripheral vasodilation, a pure vasoconstrictor, such as phenylephrine, is appropriate.

43. Anticipate bradycardia and hypotension during intubation of the tetraplegic patient.

(Scientific evidence—III; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Hypoxia or manipulation of the larynx or trachea may cause profound bradycardia or even cardiac arrest in the higher tetraplegic patient (Raw et al., 2003; Yoo et al., 2003). Atropine pretreatment may be appropriate in patients who are bradycardic prior to airway manipulation. Positive pressure ventilation can cause profound arterial hypotension as the systemic vascular resistance cannot be raised to offset the changes in intrathoracic pressure caused by intermittent positive pressure ventilation. Up to 30% of tetraplegic patients become hypotensive during induction (Yoo et al., 2003). Intravenous volume administration prior to intubation can offset the reduction in cardiac filling due to increased intrathoracic pressure with positive pressure ventilation, but additional vasopressor support is frequently required.

44. Avoid the use of succinylcholine after the first 48 hours post-cord injury.

(Scientific evidence—V; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Upregulation of acetylcholine receptors on denervated muscle, whether through an upper or lower motor neuron lesion, has long been known to put patients at risk of a potentially fatal hyperkalemic response to succinylcholine (G. A. Gronert, 1975). The time course of receptor upregulation is variable, but significant hyperkalemia is unlikely to occur in the first 48 hours following injury. In SCI patients, it is uncertain whether the risk of hyperkalemia ever resolves, and many clinicians avoid using succinylcholine indefinitely in this setting (J. A. Martyn, 2006).

45. Monitor temperature, warm IV fluids, and use a patient warming device as needed.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: The problem of unintended heat loss is common to all patients under general anesthesia and more so in patients following a spinal cord injury. Thermoregulation is impaired more severely in those with higher lesions due to cutaneous vasodilation. Intravenous fluid warmers, warm air blankets, and continuous temperature monitoring are essential to maintaining normothermia.

46. Consider the use of intraoperative spinal cord monitoring in the patient with sparing of spinal cord function.

(Scientific evidence—I/III/IV; Grade of recommendation—A; Strength of panel opinion—4)

Rationale: Tsirikos and colleagues (2004) demonstrated the utility of intraoperative somatosensory-evoked potential (SSEP) monitoring, which evaluates the integrity of the dorsal aspect of the spinal cord in patients with spinal cord injury—and in intact patients with a spinal fracture—who are undergoing spinal surgery. Recently, motor-evoked potential (MEP) monitoring has become more common during spinal surgery, particularly in surgery of the cervical spine (Calancie et al., 1998; L. Pelosi, 2002). Because MEP monitoring relies on neural pathways in the ventral portion of the cord, it is complementary to SSEP in the information it provides. Some clinicians recommend both be used (Costa et al., Epub 2006). While SSEP signals are fairly tolerant of volatile anesthetics, MEP quality is severely degraded by volatile anesthesia (Calancie et al., 1991). Intravenous anesthesia with propofol is commonly employed instead. Availability of a monitoring program may be the major determinant of its use.

Pain and Anxiety: Analgesia and Sedation

Recent literature suggests that pain may be undertreated in the emergency department. This contrasts with the Joint Commission on Accreditation of Health Care's emphasis on patients' rights to appropriate pain management. There is evidence that undersedation may allow delirium to develop in the intensive care unit, which may enhance or at least herald later development of cognitive impairment. This risk must be balanced against the risk of respiratory suppression from oversedation

of the tetraplegic person, leading to unnecessary intubation and even tracheotomy. Some patients with a stabilized spine may have very little pain, despite a major deficit, and need very little analgesia.

Following spinal cord injury, patients may immediately experience distressing pain and sensitivity relating to trauma to the cord (neuropathic pain at or below the level of the injury), the spinal nerves (at-level neuropathic pain), the spinal fracture (at-level musculoskeletal pain), or from other concomitant injuries. Painful cutaneous hypersensitivity (allodynia) is commonly seen soon after injury. In this state, pain is evoked by nonnoxious stimuli and can be very distressing. Other abnormal sensations, such as the inappropriate sense of being cold, may also be experienced. Chronic pain, which affects up to 70% of those with SCI, has been the focus of most recent research. Relatively little has been written about pain shortly after injury, including whether early treatment can affect later pain. It is known, however, that as early as 2 weeks postinjury, patients may describe severe or excruciating pain at- or below-level neuropathic pain (Siddall et al., 1999). Neuropathic pain can be difficult to measure and to categorize, and is more difficult to treat than musculoskeletal pain.

The patient with a new spinal cord injury may feel frightened and distressed, having a sense of being imprisoned in his or her body, especially if lesions are high and the loss of upper extremity function is significant. Empathy, understanding, and quality time spent with the patient is the cornerstone of care. Worsening pain should trigger a repeat neurological examination to look for underlying sinister pathology.

Concomitant brain injury compounds the difficulty of providing effective analgesia and sedation. The literature shows an increased understanding of the interaction between effective sedation and the physiological responses reflecting central nervous system blood flow, but does not yet offer standards of care in the use of specific medications. The choice of agents used should be in full knowledge of these responses. Readers are urged to consult *Guidelines for the Management of Severe Traumatic Brain Injury* (New York: Brain Trauma Foundation, www.braintrauma.org/; accessed January 16, 2008) for contemporary advice on the care of brain-injured persons.

47. Minimize the pain of allodynia. Minimize evoked pain through thoughtful patient handling.

(Scientific evidence–IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Allodynia (hypersensitivity to dynamic touch) is a type of evoked pain that is more often seen in patients with cervical injuries. It is more common in those with incomplete rather than complete neurologic lesions and may be present within minutes of injury (Siddall et al., 1999). For most people, allodynia will diminish over weeks to months. Allodynia can be minimized by remembering which dermatomes are supersensitive and avoiding brushing against them. It may be necessary to leave the sensitive parts exposed to the air. Mechanical pain will usually diminish over successive weeks as the spinal fracture heals.

48. Assess the patient's pain, preferably using a self-reported numeric rating scale.

- Minimize reliance on reports by family members, who may underestimate pain.
- If using a pain-rating scale based in part on the physiologic manifestations of stress associated with pain, recognize that some people with SCI and higher lesions may be unable to show changes in heart rate and blood pressure assessed by the pain score.
- Provide adequate analgesia unless specific contraindications exist.
- Consider short-acting sedation to allow periodic neurologic assessment.

(Scientific evidence–III/IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: The numeric rating scale is a simple verbal method of communicating pain severity. Payen et al. (2001) described a behavioral pain scale that may assist in the assessment of pain in critically ill, sedated patients, which is based on a sum score of three items reflecting facial expression, upper limb movements, and compliance with mechanical ventilation. Although tetraplegics were excluded from the study, the investigators suggested that components of the scale are independent of limb movement and may be adaptable to all people with SCI.

Neighbor et al. (2004) showed that only 48% of 540 patients treated by a trauma team received opioid analgesia within 3 hours of arrival to the emergency department. Patients at greatest risk of oligoanalgesia were those who were at the extremes of age and those who were more seriously injured (based on trauma score, GCS, and intubation). The use of standardized pain assessment tools may enable analgesia to be offered at an earlier stage of management.

49. Employ contemporary medical guidelines to manage pain and distress in ventilated patients with spinal cord injury.

(Scientific evidence–I; Grade of recommendation–A; Strength of panel opinion–5)

Rationale: Remember that patients with a cervical lesion will not show some of the physiological manifestations of pain because of autonomic disruption, yet they will be particularly distressed because of the inability to communicate either verbally or nonverbally. Guidelines from the American College of Critical Care Medicine detail assessment techniques and treatment options for pain, anxiety, delirium, and sleep disorders (Jacobi et al., 2002).

Although there are few data directly examining the management of acute pain following SCI, the effectiveness of a number of parenteral drugs has been examined in SCI neuropathic pain. These include morphine (Attal, 2002), alfentanil (Eide, 1995), lignocaine (Attal, 2000; Backonja, 1992; Finnerup, 2005), and ketamine (Eide, 1995; Kvarnstrom, 2004). During inpatient assessment of an SCI patient, parenteral drugs may be useful in the management of pain while investigations and longer term treatments are explored.

Other pharmacological approaches to neuropathic pain have been studied in patients with longer established spinal cord injury. Analgesia in patients with SCI may be augmented with the anticonvulsants gabapentin and pregabalin. Both have been shown to influence neuropathic pain in the chronic situation (Levendoglu, 2004; Siddall, 2006). Topical use of amitriptyline and ketamine has also been reported to lessen neuropathic pain in an open-label pilot study, although it did not appear to be of benefit during a blinded assessment (Lynch, 2003). Further research is needed into management of pain in the acute situation.

50. Consider the use of breath-controlled analgesia in the tetraplegic patient.

(Scientific evidence–IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Breath-controlled activation allows tetraplegic patients to control their own analgesic device (Jastrzab and Khor, 1999). Such patient-controlled devices are at least as effective as titrated intravenous injections for the relief of pain (Evans et al., 2005) and may provide a greater sense of control than would otherwise be possible.

Administration of analgesia and sedation is more challenging in the presence of a TBI. Readers are

encouraged to consult (New York: Brain Trauma Foundation, www.braintrauma.org/; accessed January 16, 2008) for detailed information.

Secondary Prevention

Patient Handling and Skin Protection

Maintaining skin integrity during the acute hospitalization phase following SCI is critical to facilitating timely transfer to a rehabilitation setting, optimizing patient outcomes, and minimizing comorbidities (complications). It is believed that the first few hours are critical because pressure changes in the subcutaneous tissues may lead to further breakdown. Although no clinical trials were found, there is supporting literature and considerable expert opinion on measures to prevent skin breakdown during the acute phase of hospitalization. For more comprehensive management of skin integrity and prevention of pressure sores, readers are referred to *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals* (Consortium for Spinal Cord Medicine, 2000). To date, no new evidence has been found to change any of the consortium's earlier guidelines. Recommendations 51–54 in this section are consistent with these guidelines and expand on the acute management phase.

Several factors contribute to the risk for skin breakdown during the initial 3 to 7 days following an acute injury. These include reduced pain sensation due to sensory loss, brain injury, or medications; immobility due to motor loss; and the use of boards, collars, traction, or other devices that immobilize injured spinal segments during transport or until definite stabilization. The areas at greatest risk for pressure ulcer formation are at the interface of support surfaces with the skin over bony prominences below the neurological level, such as the sacrum. The multiple transfers required for imaging and other diagnostic studies during the emergency and early management phase increase the risk of friction, shearing, and possible injury due to the inadvertent bumping or striking of body surfaces on equipment. Moisture and heat under the body also contribute to the risk of skin breakdown.

Several studies suggest that the early transfer of patients with a new injury to a center with expertise in SCI management is associated with a reduced incidence of skin breakdown during the early acute phase. A prospective study of 588 patients with spinal cord injury admitted to

37 centers over a 2-year period examined the incidence of 6 common complications within 60 days of injury (Aito, 2003). Those transferred within the initial 48 hours to 1 week experienced a decrease in skin breakdown on admission. A review of retrospective studies also suggests that early transfer to and treatment in a center experienced in the management of spinal cord injury resulted in a decreased risk of developing skin breakdown (Bagnall et al., 2003). Another retrospective study examined complications and length of stay in 219 patients with new injuries admitted to a center specializing in the management of SCI over a 4-year period. Both paraplegics (1.5%) and tetraplegics (1.1%) admitted within 1 week of injury were less likely to experience pressure sores than those admitted after 1 week (22.2% and 17.9%, respectively; Aung and el Masry, 1997).

51. Assess areas at risk for skin breakdown frequently.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Assess skin with each repositioning, especially over bony prominences: occiput, scapulae, sacrum/coccyx, trochanters, ankles, and heels. When a patient begins sitting upright, the skin over the ischial tuberosities is also at risk for pressure ulcer formation. Frequent assessment of skin over bony prominences and around and under any orthoses (including prophylactic compression stockings) is required to detect reddened or at-risk areas early to initiate preventive intervention (Consortium for Spinal Cord Medicine, 2000). Documentation of the initiation, evaluation, and significant findings of interventions is essential to preventing progression of ischemia.

52. Place the patient on a pressure reduction mattress or a mattress overlay, depending on the patient's condition. Use a pressure-reducing cushion when the patient is mobilized out of bed to a sitting position.

(Scientific evidence–III; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: This recommendation is based on *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals* (Consortium for Spinal Cord Medicine, 2000). The comparative study by Catz et al. (2005) factored in an economic assessment of the nursing manpower costs required to turn patients every 2 to 4 hours, suggesting that alternating pressure systems may be beneficial and cost effective.

53. Provide meticulous skin care:

- Reposition to provide pressure relief or turn at least every 2 hours while maintaining spinal precautions.
- Keep the area under the patient clean and dry and avoid temperature elevation.
- Assess nutritional status on admission and regularly thereafter.
- Inspect the skin under pressure garments and splints.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Use a foam or gel pad or a bridging device to suspend the involved area above the surface to reduce pressure (Consortium for Spinal Cord Medicine, 2000). For example, to raise the occiput of a patient in traction, place a small pad on either side of the back of the head, which will elevate the head slightly above the bed surface while maintaining alignment (Consortium for Spinal Cord Medicine, 2000). When a hard collar is in place for stabilization, the chin, ears, and occiput need frequent assessment. If a halo brace is used, pin sites need to be assessed and institutional procedures for pin care followed. If a sheepskin vest is used, periodic changes will help keep the skin under the vest clean and dry.

54. Educate the patient and family on the importance of vigilance and early intervention in maintaining skin integrity.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Teaching from the perspective of a partnership model from the outset of injury can help the patient with a new SCI acquire the knowledge and skills to achieve the best possible outcomes. Early inclusion of the patient and family in the plan of care and in the rationale for interventions can increase their level of participation and involvement and decrease feelings of helplessness and loss of control (Consortium for Spinal Cord Medicine, 2000).

Prevention and Treatment of Venous Thromboembolism

Venous thromboembolism (VTE) is a serious complication in patients with acute spinal cord injury. A recent examination of California hospital discharge data revealed that 6% of 16,240 patients admitted with spinal cord injury developed VTE (Jones et al., 2005). In the absence of prophylaxis,

at least 50% of patients with acute SCI may develop VTE. In the study, 92% of the cases developed within the first 3 months following injury. VTE was infrequent in children, but the incidence in teenagers was similar to that in adults. Additional risk factors for VTE in the study were ethnicity (African Americans had the highest rate), the presence of any comorbidity, gender (males had a higher rate than females), and intubation. This subject is discussed in depth in *Prevention of Thromboembolism in Spinal Cord Injury*, 2nd edition (Consortium for Spinal Cord Injury, 1999).

55. Apply mechanical compression devices early after injury.

(Scientific evidence—I/II; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Studies have shown that mechanical compression increases venous outflow and reduces venous stasis, but that it is relatively ineffective as a single modality in preventing VTE in very high-risk patients, such as those with acute spinal cord injury (Geerts et al., 2004). However, because compression is safe, it should be implemented in all patients with acute spinal cord injury (Winemiller et al., 1999). There is also evidence that intermittent pneumatic compression may enhance the efficacy of anticoagulant prophylaxis (Spinal Cord Injury Thromboprophylaxis Investigators, 2003; Aito et al., 2002). If trauma to the lower extremities prevents the application of stockings or devices, consider use of a foot pump. To prevent skin breakdown under these devices, review the recommendations under “Patient Handling and Skin Protection.” If implementation of anticoagulation medication or mechanical compression is delayed by more than 3 days, consider performing a duplex scan to exclude deep vein thrombosis prior to placing compression devices.

56. Begin low molecular weight heparin, or unfractionated heparin plus intermittent pneumatic compression, in all patients when primary hemostasis becomes evident. Intracranial bleeding, perispinal hematoma, or hemothorax are potential contraindications to the administration of anticoagulants, but anticoagulants may be appropriate when bleeding has stabilized.

(Scientific evidence—I/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: In the randomized, controlled clinical trial of the Spinal Cord Injury Thrombo-

prophylaxis Investigators, anticoagulant prophylaxis was initiated within 72 hours of complete spinal cord injury in 476 patients from 27 centers (Spinal Cord Injury Thromboprophylaxis Investigators, 2003). Concomitant chest/abdomen injuries were present in 24%, pelvis/lower extremity injuries in 5%, and head injuries in 2%. Patients were randomized to enoxaparin (30 mg subcutaneously every 12 hours), or unfractionated heparin (5000 U every 8 hours) plus intermittent compression. Of the 107 assessable patients (those with adequate venography), 65% in the enoxaparin group and 63% in the heparin-compression group had VTE, but pulmonary embolism was more frequent in the heparin-compression group (18% vs. 5%, $p = .03$). However, there were no deaths from fatal pulmonary emboli. Major bleeding (defined as overt bleeding that resulted in a decline of hemoglobin of 2 G/dl, in a transfusion of 2 or more units of blood, or that was perispinal, intracranial, retroperitoneal, or fatal) was observed in 3% of the enoxaparin-treated and 5% of the heparin-treated patients ($p = \text{ns}$). In summary, this study demonstrated the feasibility of initiating anticoagulant prophylaxis early in spinal cord trauma, even in the presence of other serious injuries. In addition, the use of a low molecular weight heparin—enoxaparin—was associated with a low rate of pulmonary embolism and major bleeding.

In an observational study of enoxaparin (40 mg daily) combined with compression hose, investigators found that 2 of 130 patients had deep vein thrombosis and 1 had intraspinal bleeding (Deep et al., 2001). In patients with multiple traumas, low molecular weight heparin should be considered for prophylaxis (Geerts et al., 2004). Pharmacologic prophylaxis should be held for several hours prior to elective surgery, depending on the half-life of the agent. When emergency surgery must be performed, the administration of protamine will neutralize unfractionated heparin and partially neutralize low molecular weight heparin. Low molecular weight heparin prophylaxis may be resumed 24 hours postsurgery if bleeding has been controlled.

57. Consider placing a vena cava filter only in those patients with active bleeding anticipated to persist for more than 72 hours and begin anticoagulants as soon as feasible.

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: The evidence that prophylactic vena cava filters are useful in patients with spinal cord injury is weak. Some uncontrolled studies

have supported their use (Wilson et al., 1994), while others have suggested that they are not routinely indicated (Maxwell et al., 2002). The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy specifically recommended against use of a filter as primary prophylaxis (Geerts et al., 2004). The presence of a vena cava filter is a relative contraindication to manually assisted cough (“quad coughing”) for clearance of bronchial secretions because the filter may become dislodged; use of a modified assisted cough technique with lateral compression may be a safer choice. However, in situations where other methods of thromboembolism prophylaxis cannot be used, such as in unstable patients with active bleeding, a filter should be considered (Rogers et al., 2002; Wilson et al., 1994). Permanent filters are associated with a 26% to 36% frequency of development of deep vein thrombosis on long-term follow-up (Duperier et al., 2003; PREPIC Study Group, 2005), so a temporary filter may be more appropriate (Morris et al., 2004). Because the risk of bleeding may decline during hospitalization, patients should be assessed regularly for early filter retrieval and initiation of pharmacologic prophylaxis.

Respiratory Management

58. Monitor patients closely for respiratory failure in the first days following spinal cord injury.

- Obtain baseline respiratory parameters (vital capacity, FEV1) and arterial blood gases when patients are first evaluated and at intervals until stable.
- Consider mechanical ventilation for patients with tetraplegia.
- Admit patients with complete tetraplegia and injury level at C5 or rostral to an intensive care unit.

(Scientific evidence–II/III/IV; Grade of recommendation–B; Strength of panel opinion–5)

Rationale: Patients with acute SCI are at high risk for respiratory complications. Respiratory complications that occur during the acute care phase of hospitalization are a primary determinant of both length of stay and cost of hospitalization among patients with acute tetraplegia (Winslow et al., 2002). Although respiratory complications (including ventilatory failure, atelectasis, pneumonia, and pleural effusions) are most common with C1–C4 level injuries (84% incidence), they occur in 60% or more of patients with lower injury levels at some point during acute care or rehabilitation (Jackson and Groomes, 1994). Dysphagia occurs

in at least 15% of patients with recent cervical SCI (Kirshblum et al., 1999) and may result in aspiration pneumonia.

The incidence of ventilatory failure following acute tetraplegia is as high as 74%. Moreover, 95% of patients with injury level above C5 and ASIA A status will require mechanical ventilation (Velmahos et al., 2003). Ventilatory failure may have onset with the acute injury or develop progressively over the first few days postinjury. Therefore, close monitoring of spontaneously breathing patients with new SCI is warranted to prevent acute desaturation and complications of emergent intubation. This should include frequent assessment of arterial blood gases as well as serial measurement of vital capacity, depending on level of injury.

59. Perform a tracheotomy early in the hospitalization of patients who are likely to remain ventilator dependent or to wean slowly from mechanical ventilation over an extended period of time, unless the treating center has special expertise in the use of noninvasive ventilation.

(Scientific evidence–IV/V; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Although there are potential benefits to noninvasive ventilatory support in persons with SCI (Bach, 1994a, 1994b, 2002), most patients who are expected to have prolonged or permanent ventilatory failure undergo tracheotomy. In some centers, tracheotomies are performed on 69% of patients with complete tetraplegia (Harrop et al., 2004). Patients who undergo tracheotomy are more likely to be at an advanced age, to have a higher neurological level, to have preexisting medical conditions, such as lung disease, and to have developed pneumonia. A recent systematic review and meta-analysis of early versus late tracheotomy in critically ill patients (not limited to SCI) found early tracheotomy to be associated with a reduction in length of stay in ICU and duration of mechanical ventilation, but no reduction in mortality or hospital-acquired pneumonia (Griffiths et al., 2005).

A limited number of studies have investigated ventilator management strategies for patients with recent SCI, typically beginning at the time of admission to rehabilitation and the commencement of ventilator weaning. These studies are reviewed in *Respiratory Management Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals* (Consortium for Spinal Cord Medicine, 2005). The notable finding in all the studies is that high tidal

volumes (20 ml/kg or greater) during inpatient rehabilitation were associated with improved outcomes for atelectasis and time to ventilator weaning. However, these data are limited in that they are single-center retrospective reviews with a small sample size. There are no published prospective randomized trials in mechanical ventilation for acute respiratory failure in SCI that have demonstrated improved outcome with a specific mode, rate, or tidal volume.

A randomized, controlled clinical trial of ventilator weaning has been initiated to compare high tidal volume (20 cc/kg) to low tidal volume (10 cc/kg) in an 8-week trial. The purpose is to determine if high tidal volume is associated with a decrease in atelectasis and ventilator-associated pneumonia with no increase in barotrauma or adult respiratory distress syndrome (ARDS). Inclusion criteria for this study are traumatic SCI at levels C3–C6 with ASIA A, B, or C tetraplegia; subacute admission to the hospital between 2 weeks and 6 months postinjury; complete ventilator-dependence (24 hours a day) at the time of admission to the hospital; and ages 18–55 years. The evidence-based “Guidelines for Mechanical Ventilation of the Trauma Patient” recommends that “patients not meeting ALI [acute lung injury] or ARDS criteria can be ventilated using the mode, rate, and tidal volume chosen at the treating physician’s discretion” (Nathens et al., 2005).

However, in patients with SCI who develop ALI (defined as $\text{PaO}_2/\text{FiO}_2$ ratio < 300) or ARDS (defined as $\text{PaO}_2/\text{FiO}_2$ ratio < 200), mechanical ventilation with a lower tidal volume (6 ml/kg predicted body weight) and a lower plateau pressure (30 cm of water or less) is recommended. A multicenter randomized trial ($n = 861$) documented that mechanical ventilation with a lower tidal volume (6 ml/kg vs. 12 ml/kg) and lower plateau pressure (30 cm vs. 50 cm of water or less) resulted in a decrease in hospital mortality (31% vs. 40%, $p = .007$) and an increase in the number of ventilator-free days (mean \pm SD = 12 ± 11 vs. 10 ± 11 , $p = .007$; Acute Respiratory Distress Syndrome Network, 2000). Low tidal volume ventilation should be used in all patients with ALI and ARDS because it is the only method of mechanical ventilation that, to date, has been shown to improve survival. Patients with SCI who develop ARDS should be treated with low tidal volume ventilation until their pulmonary disease resolves, as the evidence for benefit is so compelling.

Additionally, SCI-specific, single-center retrospective studies have shown shorter time to wean using a progressive ventilator-free breathing

(“T-piece”) weaning protocol for patients who are ventilator-dependent at the time of admission to rehabilitation (Peterson et al., 1994). A detailed respiratory care protocol is described in *Respiratory Management Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals* (Consortium for Spinal Cord Medicine, 2005). Patients with markedly reduced vital capacity typically require a period of weeks for complete ventilator weaning. Those who are rapidly taken off all ventilatory support but still require a high concentration of supplemental oxygen often have recurrent ventilatory failure due to atelectasis or declining lung compliance. It is typically more difficult to reinstitute ventilator use on a rehabilitation unit than to accommodate a patient who is partially ventilator-dependent on admission to rehabilitation.

60. Treat retained secretions due to expiratory muscle weakness with manually assisted coughing (“quad coughing”), pulmonary hygiene, mechanical insufflation-exsufflation, or similar expiratory aids in addition to suctioning.

(Scientific evidence–IV/V; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: Although ventilatory failure due to inspiratory muscle weakness occurs primarily with higher level cervical SCI, the consequences of expiratory muscle weakness affect patients with midthoracic injury levels and above. The primary expiratory muscles (internal intercostals and abdominals) have thoracic-level innervation, so many patients with thoracic-level paraplegia also have an ineffective cough. Disruption of sympathetic input to the bronchi with midlevel thoracic paraplegia and more rostral injuries may increase the vagally mediated production of bronchial secretions. Tracheal suctioning is often insufficient for secretion mobilization. The 4:1 predominance of left-sided atelectasis and pneumonias in the first weeks following SCI has been attributed to bronchial anatomy, with a tendency for suction catheters to not enter the left main stem bronchus (Fishburn et al., 1990). The expiratory aids listed above are effective at mobilizing bilateral bronchial secretions. Their use is described in *Respiratory Management Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals* (Consortium for Spinal Cord Medicine, 2005). In particular, mechanical insufflation-exsufflation (such as the CoughAssist device by Respironics, Inc.) produces cough flows comparable to a normal cough, can be administered via tracheostomy or mouth, and is

well tolerated and preferred by patients over deep suctioning (Bach, 1994b; Garstang et al., 2000).

61. Initiate a comprehensive protocol to prevent ventilator-associated pneumonia in patients with acute spinal cord injury who require mechanical ventilation for respiratory failure.

(Scientific evidence—I/II/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Respiratory complications are common in patients with spinal cord injuries. A recent retrospective single-institution study documented that respiratory complications occurred in 52% of patients with T1–T6 SCI (vs. 35% in T7–T12 SCI and 28% in thoracic fractures; Cotton et al., 2005). The need for intubation, the risk of pneumonia, and the risk of death were all significantly greater in patients with T1–T6 SCI. These findings suggest that high thoracic patients with SCI warrant aggressive pulmonary care, similar to that given to patients with cervical SCI. Mechanical ventilation places patients at increased risk for death and complications, including ventilator-associated pneumonia (VAP). VAP is defined as occurring more than 48 hours after the patient was intubated.

VAP preventive strategies developed for non-SCI patient populations include the following:

- Maintain a semirecumbent position (that is, elevate the head of the bed to 45 degrees) in the absence of contraindications (with care not to cause shear stress over the sacrum; Drakulovic et al., 1999).
- Interrupt sedation daily to assess readiness for ventilator weaning and extubation (Kress et al., 2000).
- Follow accepted protocols for weaning from mechanical ventilation (Dries et al., 2004).
- Use orotracheal route of intubation rather than nasotracheal.
- Change ventilator circuits only if the circuits become soiled or if a new patient begins using the equipment.
- Use closed endotracheal suction systems that are changed for each new patient or as clinically indicated.
- Use heat and moisture exchangers in the absence of contraindications.
- Change heat and moisture exchangers weekly (Dodek et al., 2004).

- Perform oral care with Chlorhexidine (Koeman et al., 2006).

Additional strategies for preventing VAP include the use of endotracheal tubes with subglottic secretions drainage and kinetic beds (Dodek et al., 2004). A group of ventilator care processes (including elevation of the head of the bed, daily “sedation vacation” and assessment of readiness to be extubated, peptic ulcer disease prophylaxis, and deep venous thrombosis prophylaxis), called the “ventilator bundle,” has resulted in a significant reduction in rates of VAP (Cocanour et al., 2006).

All of these strategies are appropriate for most patients with SCI, with the exception of the 45-degree head-of-bed elevation. This may be contraindicated due to the risk of pressure ulcer formation from skin shearing; however, frequent repositioning may minimize skin problems when the head of the bed is elevated.

Genitourinary Tract

62. Place an indwelling urinary catheter as part of the initial patient assessment unless contraindicated. If contraindicated, use emergent suprapubic drainage instead.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Persons with spinal cord injury have a neurologic loss of the ability to void. In the immediate period after complete SCI, the lower genitourinary tract exhibits loss of all reflex activity, such that urinary retention is common, even to very high volumes. Even in those with incomplete injuries, urinary retention is common. Therefore, transurethral bladder catheterization should be initiated no later than the emergency department, and ideally when IV fluids are initiated. An indwelling bladder catheter offers the advantages of measuring urine output accurately and preventing overfilling of the bladder.

Urethral injury should be suspected after pelvic fracture, traumatic catheterization, or penetrating injury near the urethra. Features of urethral injury after SCI include hematuria, blood at the meatus, or a high-riding prostate gland. Instrumentation—such as attempting placement of a urethral catheter—can further exacerbate urethral injury, which can lead to long-term complications, such as stricture or difficulty voiding. In these cases, a urologist should be immediately consulted to determine the optimal treatment to establish bladder drainage and treat the urethral injury.

63. Leave indwelling urinary catheters in place at least until the patient is hemodynamically stable and strict attention to fluid status is no longer needed.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Spinal cord injury results in a sudden loss of autonomic control, so vasoconstriction and dilatation no longer maintain venous return. The recumbent position and loss of muscle pump contribute to this mechanism, making third spacing of fluid nearly universal. Hypotension results from the loss of sympathetic control and is commonly treated with fluid resuscitation and/or vasopressors. Oliguria is common in the early period, likely as a result of third spacing, so patients are commonly many liters fluid positive. Hormonal changes have also been described, although the onset of these changes is unclear. Therefore, maintenance of an indwelling urethral catheter is preferable during the early period since it allows for more precise monitoring of urinary output than other bladder management techniques.

Chronic neurogenic bladder dysfunction due to SCI often can be managed with techniques that do not require an indwelling urinary catheter. Readers are referred to *Bladder Management for Adults with Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Providers* (Consortium for Spinal Cord Medicine, 2006). Some of these methods may reduce the rate of significant bacteriuria (Johnson et al., 2006). However, there is insufficient evidence to determine optimal timing for transition from the indwelling catheter to other methods, such as intermittent catheterization of the bladder.

64. Priapism is usually self-limited in acute SCI and does not require treatment. There is no evidence to support avoidance of a urethral catheter in the presence of priapism secondary to acute SCI.

(Scientific evidence–II; Grade of recommendation–B; Strength of panel opinion–5)

Rationale: Priapism is a common finding in the prehospital setting and during the early period after SCI. Recognized as a sign of spinal cord injury, it is commonly regarded as self-limited without need for treatment. Textbooks have previously recommended avoidance of transurethral catheterization, but few data support this recommendation. One study followed four patients with priapism lasting up to 5 hours. The authors concluded that conservative management provides

good short- and long-term outcomes (Gordon et al., 2005). Consider consultation with a urologist if priapism is prolonged.

Gastrointestinal Tract

65. Initiate stress ulcer prophylaxis.

(Scientific evidence–I/III/IV; Grade of recommendation–A; Strength of panel opinion–5)

Rationale: Patients with acute SCI are at high risk of gastrointestinal (GI) bleeding for 4 weeks or until other risk factors for the bleeding resolve, whichever is longer. Patients should not be placed on prophylaxis longer than 4 weeks unless other risk factors are present.

Two strong independent risk factors for GI bleeding were identified in critically ill patients ($n = 2,252$): respiratory failure (odds ratio = 15.6) and coagulopathy (odds ratio = 4.3; Cook et al., 1994). In a study of patients requiring mechanical ventilation for at least 48 hours ($n = 1,077$), renal failure was independently associated with an increased risk of clinically important GI bleeding, whereas enteral nutrition and stress ulcer prophylaxis with ranitidine conferred protection (Cook et al., 1999). Gastrointestinal stress ulceration is a well-recognized complication of trauma, with SCI being an independent risk factor for GI bleeding among trauma patients. The incidence of GI bleeding is relatively low (2.5%–8%), however, with nearly universal use of prophylaxis (Albert et al., 1991). Patients with cervical complete injuries have consistently shown a greater risk of GI bleeding (Kiwinski, 1986; Walters and Silver, 1986), and animal data have suggested excessive gastric acid production associated with excessive vagal tone in this group.

Histamine H₂-receptor antagonists (H₂RAs) have demonstrated greater efficacy than the cytoprotective agent sucralfate, which does not alter the gastric pH. A multicenter, randomized, blinded, placebo-controlled trial compared sucralfate (1 g q6h) with the H₂-receptor antagonist ranitidine (50 mg q8h) for the prevention of GI bleeding in 1,200 patients who required mechanical ventilation. Ranitidine use was associated with a decrease in GI bleeding (relative risk = 0.44; 95% confidence interval = 0.21, 0.92; $p = .02$) and no increase in the incidence of ventilator-associated pneumonia (Cook et al., 1998).

Proton pump inhibitors (PPIs) achieve a more rapid and sustained increase in gastric pH. H₂RAs block only one of three pathways in acid secretion and provide less potent acid suppression than the PPIs, which block the final common

pathway in acid secretion. In addition, tolerance that occurs with H2RAs does not occur with PPIs. H2RAs dosing, but not PPI dosing, must be adjusted for patients with renal dysfunction (Welage, 2005). Furthermore, immediate-release PPI suspension was documented to be as effective in preventing upper GI bleeding and more effective than intravenous H2RA in maintaining gastric pH of > 4 in critically ill patients (n = 359) who required mechanical ventilation and had at least one additional risk factor for upper GI bleeding (Conrad et al., 2005). Therefore, based on the evidence to date, both H2RAs and PPIs are safe agents to use for acid suppression to prevent stress-related mucosal disease in patients with acute SCI.

However, indiscriminate use of PPIs has been raised as a possible cause of the increasing rate of *Clostridium difficile* infection in hospitals and the community, so prolonged prophylaxis without clear indication does not appear to be benign. Most peptic ulcerations occur within 4 weeks of injury, with the risk of ulceration specific to SCI diminishing thereafter. Other absolute risk factors (such as premorbid peptic ulcers, prolonged mechanical ventilation, or coagulopathy) or relative risk factors (such as liver failure, burns, or ongoing use of corticosteroids) may prolong the need for prophylaxis (Albert et al., 1991; Dial et al., 2005; Kiwerski, 1986).

66. Evaluate swallowing function prior to oral feeding in any acute SCI patient with cervical spinal cord injury, halo fixation, cervical spine surgery, prolonged intubation, tracheotomy, or concomitant TBI.

(Scientific evidence—I/II/III/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Dysphagia is present in 17% to 41% of patients with tetraplegia, as determined by diagnostic studies performed at varying times between the acute period and discharge from rehabilitation (Abel et al., 2004; Kirshblum et al., 1999; Wolf and Meiners, 2003). Rates in the acute period may be higher. Risk factors for dysphagia in these studies include tracheotomy, presence of a halo orthosis, anterior cervical spine surgery, and higher neurological level. Studies performed in healthy volunteers indicate that changes in swallowing function are common with cervical collars or halo vest braces (Morishima et al., 2005; Stambolis et al., 2003). In patients without SCI undergoing anterior cervical discectomy and fusion, dysphagia is common and has been attributed to soft-tissue swelling or vocal cord paresis (Frempong-

Boadu et al., 2002). Concomitant brain injury is common in patients with SCI and is also a well-recognized risk factor for dysphagia.

Bowel Care

67. Initiate a bowel program as recommended in the clinical practice guideline *Neurogenic Bowel Management in Adults with Spinal Cord Injury*.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Loss of colonic motility early after SCI is well recognized. Other aspects of trauma, surgery, and medications may all contribute to poor gut motility and even ileus in the newly injured patient. Bowel distention and inadequate evacuation can lead to nausea and vomiting, high gastric residuals, anorexia, poor lung expansion, and inadequate venous return. Therefore, attention to bowel evacuation early after injury can prevent complications and reduce acute care length of stay.

Once patients are fed enterally, bowel movements should be expected and facilitated as part of routine care. The goal for patients with neurogenic bowel dysfunction is to have one scheduled bowel movement per day, with use of oral medications, suppositories, and digital stimulation as needed to trigger the bowel movement. The components of a bowel program are described in the clinical practice guideline *Neurogenic Bowel Management in Adults with Spinal Cord Injury* (Consortium for Spinal Cord Medicine, 1998). The procedures for the bowel program are chosen based on the presence or absence of the bulbocavernosus reflex (BCR), which is an indicator of upper motor neuron versus lower motor neuron bowel dysfunction.

Most patients lose sacral reflexes in the early period. Timing of the return of these reflexes and the ability to use them for functional bowel evacuation is variable, but can be monitored via the BCR. If the BCR is initially absent in a patient who is anticipated to have upper motor neuron bowel dysfunction (i.e., injury level above the cauda equina or terminal part of the conus), it should be rechecked daily for at least the first few days, and bowel care should be modified accordingly if it returns. A bowel program based on these guidelines may not be possible in some patients because of concomitant injuries, prohibitions against turning, or other complications. In these cases, as well as in the setting of diarrhea, a rectal tube may be a useful temporary measure until a bowel program can be initiated.

Nutrition

68. Provide appropriate nutrition when resuscitation has been completed and there is no evidence of ongoing shock or hypoperfusion.

- Use enteral nutrition rather than parenteral nutrition.
- Feed a standard, polymeric enteral formula initiated within 24 to 48 hours after admission, using the semirecumbent position when possible to prevent aspiration.
- Determine the caloric requirements for nutritional support in acute SCI using a 30-minute energy expenditure measurement by indirect calorimetry (metabolic cart).

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Nutritional support is necessary in all trauma patients, and early enteral nutritional support in trauma has been associated with improved outcomes. Nutrition is recognized as a necessary intervention to prevent complications and optimize healing in multiple trauma, burn, and brain injuries (AANS and CNS, 2002). In these populations, initiation of feeding within 48 to 72 hours can reduce complications, improve outcomes, and decrease length of stay. In spinal cord injury in particular, however, such improvement has not been clearly demonstrated. Enteral feeding has been shown to decrease gastric stress ulceration in an acute SCI population (Kuric et al., 1989), and even parenteral nutrition appears to decrease ulceration in persons with SCI. However, in a randomized pilot study of acute traumatic tetraplegia comparing initiation of enteral feeding less than 72 hours after injury and more than 120 hours postinjury, early feeding demonstrated no benefit with respect to infection, length of mechanical ventilation, or overall length of stay (Dvorak et al., 2004). Parenteral nutrition has not been similarly studied.

Enteral nutrition, compared with parenteral nutrition, is associated with a significant decrease in infectious complications and with a lower incidence of hyperglycemia (Gramlich et al., 2004; Heyland et al., 2003). Enrichment with glutamine, considered an option for multiple-trauma patients (Garcia-de-Lorenzo, 2003), has not been studied in patients with SCI. Although a semirecumbent feeding position reduces the risk of VAP, this position frequently places excessive shear on the skin over the sacrum unless a specialized mattress is used. Patients with significant neurological deficits need close monitoring of skin integrity if the semirecumbent position is used.

The 24-hour urine urea nitrogen (UUN) has been shown to be an unreliable measure of nutrition in acute SCI (Rodriguez et al., 1997). The negative nitrogen balance seen acutely in most patients likely reflects an obligatory loss of muscle mass associated with paralysis, much as the obligatory loss of calcium from bone. Indirect calorimetry has shown that traditional calculated caloric needs result in overfeeding. The actual caloric needs of patients in acute rehabilitation are 45% to 90% of calculated values and are lower among those with tetraplegia compared with those with paraplegia (Cox et al., 1985). However, in the acute phase, these differences are less pronounced. Adjusting the standard equations for SCI may provide an adequate estimate for the initial phase (Dvorak et al., 2004; Rodriguez et al., 1997). Predicted energy expenditure calculations should use an activity factor of 1 and a stress factor of 1.2 to 1.4 in persons with motor-complete SCI, regardless of level of injury (Rodriguez et al., 1997).

Glycemic Control

69. Maintain normoglycemia in critically ill mechanically ventilated patients.

(Scientific evidence—I/II/III/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Intensive insulin therapy (maintenance of blood glucose between 80 and 110 mg/dl) was associated with a significant reduction in ICU mortality compared with conventional treatment (infusion of insulin only if blood glucose level exceeded 215 mg/dl and maintenance of glucose between 180 and 200 mg/dl) in adult patients receiving mechanical ventilation admitted to a surgical ICU in a single-institution prospective randomized study (Van den Berghe et al., 2001). At 12 months, with a total of 1,548 patients enrolled, intensive insulin therapy reduced mortality during intensive care from 8% with conventional treatment to 5% ($p < .04$, with adjustment for sequential analyses). The benefit of intensive insulin therapy was seen in its effect on mortality among patients who remained in the intensive care unit for more than 5 days (20% with conventional treatment, as compared with 11% with intensive insulin therapy, $p = .005$). The greatest reduction in mortality involved deaths due to multiple organ failure with a proven septic focus. Intensive insulin therapy also reduced overall in-hospital mortality by 34%, bloodstream infections by 46%, acute renal failure requiring dialysis or hemofiltration by 41%, the median number of red-cell transfusions by 50%, and critical illness polyneuropathy by 44%. Patients receiving inten-

sive therapy were less likely to require prolonged mechanical ventilation and intensive care. Most of the patients in this study were cardiac surgery patients (63%); 4% were multiple-trauma or severe burn patients; and 4% had neurologic disease, cerebral trauma, or brain surgery. This study is relevant, however, because patients with cervical spine injuries above C5 and complete tetraplegia usually require mechanical ventilatory support.

In a second prospective randomized trial in medical ICU patients ($n = 1,200$) with the same experimental study design, ICU and hospital mortality was reduced only in those patients who stayed in the ICU for 3 or more days (ICU mortality 38% vs. 31%, $p = .05$; hospital mortality 53% vs. 43%, $p = .009$); no effect on mortality was identified in the intent-to-treat group (Van den Berghe et al., 2006). Morbidity was significantly reduced in the intensive insulin therapy group, especially after newly acquired kidney injury and with earlier weaning from mechanical ventilation. (Among 433 patients who stayed in the ICU for less than 3 days, mortality was greater among those receiving intensive insulin therapy.)

A number of prospective cohort studies have documented that hyperglycemia is associated with adverse outcome in trauma, including increased health-care-associated infections (VAP, surgical site infection, catheter-related bacteremia) and hospital mortality. In fact, some studies suggest that the relation of hyperglycemia and mortality is more pronounced in trauma patients than in other surgical ICU patients. Hyperglycemia at admission has been identified as an independent predictor of adverse outcome and increased infection in trauma patients (Bochicchio et al., 2005a, 2005b; Collier et al., 2005; Garber et al., 2004; Jeremitsky et al., 2005; Sala et al., 1999; Sung et al., 2005; Thorell et al., 2004; Vogelzang et al., 2006; Yendamuri et al., 2003).

Prognosis for Neurological Recovery

70. Within the first 72 hours, use the clinical neurological assessment as described by the International Standards for Neurological Classification of SCI to determine the preliminary prognosis for neurological recovery.

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Neurologic recovery commonly occurs following SCI, usually in the level adjacent to the lowest normal level, and below the injury level in those with incomplete injuries. Substantial effort has been made to identify reliable predictors of neurological recovery following SCI (Kirshblum

and O'Connor, 1998), and there is evidence that the early clinical motor and sensory examinations are highly predictive of neurological status at long-term follow-up. Early determination of neurological prognosis facilitates clinical decision making in a number of areas. Patients and families frequently request information on prognosis soon after injury. The expected level of disability is important in planning for appropriate rehabilitation services and postdischarge care needs. Functional outcomes, such as independence in self-care and ambulation are directly related to motor outcomes (Consortium for Spinal Cord Medicine, 1999). Motor and sensory scores from as early as the first 4 days postinjury predict the degree of disability at 2 years postinjury (Saboe et al., 1997). Patients who are expected to have significant residual motor, bladder, bowel, and/or sexual impairment may benefit the most from referral to regional SCI rehabilitation centers, while those anticipated to have only minimal motor deficits may be successfully managed at less specialized centers. Decisions on the medical and surgical management of some associated injuries, such as complex lower limb fractures, may also be guided by the likelihood that the patient will recover ambulation.

A number of investigators have determined the ability to predict long-term motor recovery based on early examinations. Although earlier investigations focused on the examination at 3 to 7 days postinjury, it appears that the examination performed during the first day postinjury also has high predictive value in identifying complete (ASIA A) injuries, provided that distracting factors are absent. Burns et al. (2003) reported that 7% of patients with reliable initial exams showing ASIA A converted to ASIA B after 1 year, and none converted to motor-incomplete status. Of initially ASIA A patients who had factors affecting the reliability of the examination, such as mechanical ventilation or intoxication, 17% converted to ASIA B and 13% converted to ASIA C status. Patients with initial ASIA B status have significantly greater potential for neurological and functional recovery, with 54% converting to ASIA C or D by one-year follow-up (Marino et al., 1999). Neurological recovery is more common and of greater magnitude in patients with initial ASIA C or D status. Nearly all initially ASIA C patients under 50 years of age and all initially ASIA D patients are expected to be ambulatory, with or without assistive devices, on discharge from rehabilitation (Burns et al., 1997). Most motor recovery occurs during the first 6 months postinjury, but clinically significant strength gains can occur for up to 2 years after injury.

71. If the clinical exam is unreliable, MRI findings or electrodiagnostic studies may be useful for determining prognosis.

(Scientific evidence—I/III/IV; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Diagnostic testing can provide additional information on neurological classification or prognosis in patients with potentially unreliable clinical examinations. Cord hemorrhage on both early and postoperative MRI is associated with ASIA A injuries, and thus a poor prognosis for motor recovery (Flanders et al., 1996; Schaefer et al., 1992; Selden et al., 1999). Although the clinical examination is the single best predictor of neurological improvement (Selden et al., 1999), MRI findings of hemorrhage and edema modestly improve the ability to predict motor recovery at 1 year (Flanders et al., 1996). In another MRI study of SCI, the presence of extensive edema was found to provide a poorer prognosis; hemorrhage length of 4 mm or longer had a poor prognosis for neurological recovery at long-term follow-up (Boldin et al., 2006). Calancie and colleagues (2004) used surface electromyography to measure lower limb deep tendon reflexes in the first weeks postinjury. They found that the absence of large-amplitude responses and of a crossed adductor response was predictive of motor-complete status at follow-up. Findings on early somatosensory-evoked potentials predict ambulation recovery, but they are no more accurate than the clinical examination of a cooperative and communicative patient (Curt and Dietz, 1997; Jacobs et al., 1995). Compared with clinical neurological assessment, motor-evoked potentials provide no prognostic information on the likelihood of recovering strength in initially paralyzed muscles (Macdonell and Donnan, 1995).

Rehabilitation Intervention

72. Develop protocols that allow rehabilitation specialists to become involved early in the management of persons with SCI, immediately following injury during the acute hospitalization phase.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Although the initial phase of treatment of newly injured patients centers on medical interventions to resuscitate and stabilize, this phase can last several days. During this time, the patient is often moved from the emergency room to the operating room to the ICU or appropriate unit.

Many clinicians are involved with the care of the patient and are developing plans for the rest of the hospitalization, if not longer. Evaluations by various members of the multidisciplinary team using standardized guidelines should assess the type, level, and severity of the injury to aid in the development of a comprehensive plan of care.

Pathways, standing orders, and protocols for patients with SCI that allow for the early evaluation, assessment, and treatment by physical therapists, occupational therapists, rehabilitation nurses, and speech and language pathologists immediately trigger the involvement of these and other rehabilitation team members. Early intervention by rehabilitation specialists may shorten length of stay during the acute hospitalization phase by preventing secondary complications and moving the patient more quickly toward discharge to the next level of care.

73. Prescribe interventions that will assist the recovery of persons with SCI, including preventive measures against possible secondary complications. Educate patients and families about the rehabilitation process and encourage their participation in discharge planning discussions.

(Scientific evidence—NA; Grade of recommendation—NA; Strength of panel opinion—5)

Rationale: Individuals with SCI present with deficits in a variety of areas. The rehabilitation team is trained to help improve function and prevent further deterioration in many of these areas. Especially vital is to initiate range of motion exercises of all joints within the first week after injury and to continue them during the acute phase. Stretching of the joints should begin as soon as either a loss of range of motion or an increase in tone is detected across the joint.

Generally, the rehabilitation discipline listed below is responsible for implementing the following types of interventions. Specific responsibilities will differ among staffs in different facilities; for example, initial prescription of an appropriate wheelchair may be undertaken by a rehabilitation nurse, a physiotherapist, or occupational therapist.

Physical Therapy

- Range of motion and strengthening exercises.
- Pulmonary interventions, such as pulmonary hygiene, percussion, vibration, suctioning, postural drainage, mobilization, training of accessory muscles and/or glossopharyngeal pistoning breathing, cough, and deep breathing exercises; working with respiratory therapists and nursing staff.

- Seating and positioning.
- Mobilization, including bed mobility, transfer training, locomotion.
- Lower extremity splinting.
- Patient, family, and caregiver education.

Occupational Therapy

- Range of motion, strengthening, and stretching exercises.
- Upper extremity splint fabrication.
- Positioning and seating.
- Retraining for activities of daily living.
- Edema management.
- Patient, family, and caregiver education.
- Assessment of swallowing.

Speech and Language Pathology

- Functional and/or augmentative communication.
- Assessment of swallowing.
- Assessment of cognitive and/or language deficits from concomitant TBI.
- Patient, family, and caregiver education.

Patients with SCI are subject to a host of secondary complications, including pressure ulcers, respiratory complications, orthostatic hypotension, joint contractures, muscle weakness, venous thromboembolism, and upper limb pain. Many of these conditions can be prevented. Rehabilitation specialists, with their expertise in the use of specialized interventions, such as positioning, early mobilization and functional mobility training, splinting, assessment for specialized equipment, and patient education, can be instrumental in this regard. Moreover, early treatment may enhance recovery and functional independence. However, evidence on the incremental risks and benefits of each specific intervention is lacking, and research in this area is needed.

Early education of patients, families, and caregivers allows them to begin to assist with care as early as possible. Rehabilitation specialists can inform patients and their families about expected outcomes, discharge recommendations, aspects of care in which they can participate, and the goals of therapy. Education helps patients to recognize the potential signs and symptoms of

complications, allows them to make thoughtful decisions about their care, and assists them in their own psychological adjustment process following SCI.

As early as possible, rehabilitation team members should make recommendations about the appropriate subsequent level of care to assist both the patient and the care team with the transition. Some patients who show rapid neurologic improvement or who have minimal deficits may be able to be discharged to home within a few days of injury. For these patients, comprehensive discharge planning should begin as early as possible. Home accessibility, required medical equipment, and availability of follow-up services, such as outpatient therapy, should all be assessed. However, after the period of acute care, most patients with SCI will benefit from transfer to a comprehensive inpatient rehabilitation unit with expertise in SCI. In these cases, it is recommended that team members begin to define some components of the postdischarge plan, including the likely residence following discharge and the availability of family members and friends who can provide assistance if necessary. The expected functional outcomes and equipment needs based on neurological level are described in Outcomes Following Traumatic Spinal Cord Injury: Clinical Practice Guidelines for Health-Care Professionals (Consortium for Spinal Cord Medicine, 1999).

74. Use nonpharmacologic and pharmacologic interventions for orthostatic hypotension as needed. Mobilize the patient out of bed to a seated position once there is medical and spinal stability. Develop an appropriate program for out-of-bed sitting. Limit in-bed and out-of bed semireclined sitting, as this often produces excessive skin shear and predisposes to pressure ulcer formation.

(Scientific evidence—III; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Training to restore independent mobility, whether ambulation or wheelchair mobility, requires the patient to tolerate an upright body position. Secondary benefits of mobilization to an upright position include limitation of deconditioning, a reduction in respiratory complications, a reduction in pressure over the sacrum, and the psychological reward of interacting with the environment from an upright position. The timing of mobilization following injury needs to be based on the patient's medical status and spinal stability.

Orthostatic hypotension is common during the first days following injury, due to loss of sympathetically mediated vascular tone with injuries

at a thoracic or cervical level plus reduced venous return from dependent lower limbs secondary to muscle paralysis. Nonpharmacologic interventions for orthostatic hypotension include lower limb compression with graduated elastic stockings and elastic wraps, abdominal binders (especially with cervical and high-level motor-complete injuries), and gradual attainment of an upright position. With more severe orthostatic hypotension, pharmacologic therapy is often also needed. Ideally, patients will be mobilized to a high-back wheelchair that can be reclined if symptomatic hypotension develops, with the goal of attaining fully upright sitting as tolerated.

Skin protection is optimized if the patient sits fully upright in a wheelchair with a specialized cushion for pressure reduction. It is necessary to perform weight shifting to periodically relieve pressure on the skin. Patients should be instructed as early as possible to perform weight shifting independently if able. Staff and caregivers should be instructed on how to perform pressure relief if the patient is unable to do so. The bedside chairs typically provided in ICU environments are not preferred, since the semireclined back may place excessive shear across the sacral skin, the seat cushion may not provide adequate pressure relief, and postural support is often insufficient. Sitting upright in bed with the head of the bed elevated also produces shear across the sacrum, and pressure-reducing mattresses are less effective for this body position (Goetz et al., 2002).

Psychosocial and Family Issues

Although no studies were found that were conducted during the initial 3 to 7 days following spinal cord injury, qualitative and retrospective studies give some insight into what patients experience during their early recovery. Initially, patients and family members may experience feelings of gratitude at having survived the traumatic event. Then, individuals who suffered an acute spinal cord injury describe a plethora of feelings, including uncertainty, loss, hope, grief, anxiety, depression, despair, helplessness, loss of control, powerlessness, and suicidal ideation.

Several psychosocial issues may emerge within this context: (1) grief and denial reactions, (2) major depression, and (3) decisions to remove life support. Although often applied informally to the SCI adjustment process, traditional grief models (Kübler-Ross, 1969) do not appear valid generally (Stroebe and Schut, 1999; Wortman and Silver, 1989) or in relation to SCI (Elliott and Frank, 1996; Trieschmann, 1988). Consistent with other types

of severe loss, the modal response of people with SCI is expected to be resilience, not depression (Bonanno, 2004). More evidence-based models describe a usual grief process as oscillation between intrusive feelings of sadness, despair, and anxiety versus feelings of numbness, optimism, and gratitude (Stroebe and Schut, 1999). These oscillations can give rise to concerns that the patient is either depressed or in denial, depending on which phase is more prominent. Additionally, it appears that having “positive illusions” and exaggerated optimism regarding health is normative, healthy, and probably protective (Taylor and Brown, 1994). Families and staff may need to be educated about common myths and erroneous assumptions, such as that “depression is necessary” or “the absence of depression (denial) is pathologic.” Although it is often necessary to give diagnostic and prognostic information, it seems prudent to give this information in a way that allows the person to maintain hope.

No study has examined major depression prior to inpatient rehabilitation for SCI. The best evidence we have from inpatient rehabilitation studies using structured diagnostic assessments is that major depressive disorder (MDD) is expected in about 20% to 30% of people with SCI (Fullerton et al., 1981; Judd and Brown, 1987). When significant symptoms of depression emerge soon after SCI, there is a high (almost 50%) spontaneous remission rate within 1 week (Judd et al., 1989). Therefore, MDD should be treated aggressively, but preferably only if the person meets standard Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV) criteria, including the temporal criterion. Although no studies have proven that antidepressant medications are effective for MDD in people with SCI, it seems prudent to treat MDD medically as outlined in *Depression Following Spinal Cord Injury: A Clinical Practice Guideline for Primary Care Physicians* (Consortium for Spinal Cord Medicine, 1998). Many of the recommendations in this guideline also apply in the acute management phase. These include assessment, early detection, and treatment; a supportive-educational approach; use of pharmacologic agents if indicated; referral to team members, such as psychologists, social workers, clergy members, or case managers, to address social and environmental issues; and evaluation of the treatment plan.

Early interventions focus on factors unique to the intensive and acute care environment and on recent research findings regarding the feelings and experiences a person with a new spinal cord injury may encounter while in the acute care setting (Lohne and Severinsson, 2004a, 2004b, 2005; Martz et al., 2005; Sullivan, 2001). Suggested

interventions underscore the need to recognize and manage uncertainty, foster realistic hope, begin the adjustment to losses, bolster interpersonal support, foster independence with assisted technology, form a partnership with the health-care team, mobilize individual and family resources, assess learning styles, provide culturally sensitive care, and detect and deal with suicidal ideation and requests for withdrawal of treatment.

75. Assess mental health in general and possible risk for psychosocial problems after admission and throughout acute care stay. Involve members of the health-care team as needed. Pay particular attention to the following factors:

- Current major depression, acute stress disorder/posttraumatic stress disorder (PTSD), or substance intoxication and withdrawal.
- Social support network (or lack thereof).
- Cognitive functioning and learning style.
- Personal and cultural preferences in coping style and social support.
- Concurrent life stressors.
- Concomitant health problems, medical conditions, medications, and history of TBI.
- History of mental illness, including major depression, PTSD, substance abuse.
- Use of psychiatric medications.

(Scientific evidence–NA; Grade of recommendation–NA; Strength of panel opinion–5)

Rationale: Ongoing assessments of mental health status are essential to developing an effective intervention plan. Every assessment should examine both individual and family coping skills and level of support. A history of previous mental health problems, such as depression, is the best indicator of risk for the development of similar problems in response to spinal cord injury (Consortium for Spinal Cord Medicine, 1998). Assessments of culturally relevant nuances help to ensure that care will be culturally competent (Sharma and Smith, 2002).

76. Foster effective coping strategies, health-promotion behaviors, and independence through a variety of ongoing interventions.

- Use assistive devices, such as head-controlled call bells, bed controls, prism glasses, and communication boards.
- Acknowledge that feelings of gratitude, uncertainty, loss, and helplessness may be present simultaneously.
- Provide medical and prognostic information matter-of-factly, yet at the same time leave room for hope.
- Respect expressions of hope. Avoid direct confrontations of denial concerning probable implications of the injury.
- Help the patient and family to identify effective coping strategies that have aided them in the past.
- Develop a partnership of patient, family, and health-care team to promote involvement in the treatment plan and optimize patient outcomes.

(Scientific evidence–III/IV; Grade of recommendation–C; Strength of panel opinion–5)

Rationale: People with a new spinal cord injury often experience a range of thoughts and feelings at the same time: gratitude for having survived a traumatic event, uncertainty about what the future might hold, a sense of loss over being unable to move parts of the body and what that might mean, and thoughts of helplessness and loss of control when unable to do things for themselves (Sullivan, 2001).

Several qualitative studies suggest that what was once thought to be denial is actually an expression of hope, which may be unrealistic at first. Almost universally, people with a new spinal cord injury state that they realized within a few minutes what had happened to them. And for the first several days, weeks, months, and even years following injury, almost everyone maintains that they will walk again. Over time, with incremental progress and recovery, this hope becomes more realistic (Laskowski and Morse, 1993; Lohne and Severinsson, 2004a, 2004b; Morse and Doberneck, 1995). Studies indicate that feelings of hope assist with a future orientation and foster the ability to move forward through the recovery process. Depression, on the other hand, diminishes the future orientation and impedes the forward moving momentum (Martz et al., 2005). Therefore, information about the injury, the possible sequelae, and the treatment plan should be discussed with the patient and family

in a matter-of-fact manner. Care should be taken to ensure that prognostic and quality-of-life information is based on the evidence (Bach and Tilton, 1994; Hall et al., 1999; Patterson et al., 1993).

When appropriate, enlist members of the health-care team to help the individual and family recall previous difficulties and identify the coping skills that helped them through the earlier crisis. These same coping skills may be beneficial in the current circumstance. Mobilization of both the individual's and the family's resources will be necessary to move through the acute and rehabilitation phases of recovery (Dewar, 2000; Kennedy et al., 2003).

To ensure that the patient feels like an active participant in the development of the treatment plan, incorporate the patient's values, beliefs, experiences, and goals into the planning process as much as possible (Scanlon, 2003). For patients who are unable to communicate, the health-care team should ask close family members and friends to create a picture of the whole person by talking about the patient's values and beliefs. Identifying what factors make life meaningful for the person with a new SCI and initiating an ongoing dialogue about the extent to which this quality of life could be maintained contribute to feelings of hope. Involving all members of the health-care team or just a few with whom the patient has established a special connection can facilitate the discussion (Lohne and Severinsson, 2005).

A philosophical shift in the role of the health-care team with patients and families has occurred in the past decade. Team members now provide interpersonal support while working with the patient and family to establish an individualized plan of care from the outset that is based on meaningful and realistic goals (Heenan and Piotrowski, 2000).

77. Detect suicidal ideation and requests for assisted suicide. Take treatment refusals and requests for withdrawal of treatment very seriously.

- Acknowledge the patient's suffering.
- Assess for and treat any underlying depression, substance abuse, or other chronic condition.
- Determine the patient's decision-making capacity.
- Identify patient needs jointly and establish a plan of care.
- Ensure informed consent.
- Consult the institution's ethics committee when appropriate.

- Consult legal counsel if the conflict continues or if there is any uncertainty regarding the patient's request.

(Scientific evidence—I/III/IV/V; Grade of recommendation—A; Strength of panel opinion—5)

Rationale: Occasionally in the acute recovery phase, patients may feel unable to cope with what they are experiencing. For this reason, it is critical that health-team members be alert to suicidal thoughts and open to discussing them with the patient. Respond honestly and sincerely if such thoughts are expressed. Maintain an ongoing dialogue about the recovery process and the likelihood of returning to a meaningful life after SCI. Emphasize that most people with SCI experience a high quality of life and feel glad to be alive with the right amount of interpersonal support, the right resources and equipment, and adequate access throughout their environment (Bach and Tilton, 1994; Hall et al., 1999). Acknowledgment of the patient's suffering is an important step in establishing trust in the relationship. Allowing the patient to freely express his or her feelings of grief, sorrow, loss, guilt, frustration, helplessness, loneliness, and dependency can lead to new feelings of respect for self and others and to a greater sense of wholeness.

Although suicidal ideation and completed suicide are somewhat more prevalent among people with SCI (15% and 59/100,000, respectively), they are still rare occurrences and should not be considered a normal response to SCI (Bombardier et al., 2004; Charlifue and Gerhart, 1991; Krause et al., 2000). Health-care providers must balance a number of factors when considering an overt refusal or a request for withdrawal of treatment (Kraft, 1999), including the patient's right to self-determination and the health-care provider's duty to benefit the patient and prevent harm. The competing obligations to do good, prevent harm, foster patient decision making, and ensure the fair use of resources are paramount.

Patterson and colleagues (1993) argued that personal bias and a lack of information among acute medical staff concerning quality of life after tetraplegia influenced the decision to withdraw life support soon after cervical SCI. In the case of high tetraplegia, there is evidence to suggest that emergency personnel significantly underestimate the potential quality of life for these patients (Gerhart et al., 1994). Whereas only 18% of emergency department personnel thought they would be glad to be alive after high tetraplegia, survey data show that greater than 90% of people with high tetraplegia report being glad to be alive. Therefore, any decision to withdraw life support soon after SCI should be scrutinized carefully.

Social workers, psychologists, psychiatrists, or other mental health professionals on the team should evaluate the patient for depression and feelings of hopelessness and make recommendations for intervention (Kishi et al., 2001). When evaluating a request for withdrawal of life support, major depression needs to be ruled out as this condition may impact the capacity for making decisions. It may be necessary to treat major depression first to see if the wish to die resolves (Leeman, 1999).

If, after pain, depression, and other acute or chronic conditions have been adequately treated the patient persists in seeking withdrawal of life support, the patient's ability to make an informed choice must be reassessed. The decision-making capacity involves the ability to (1) understand information about treatment options and their consequences, (2) determine how the information applies in the current situation and weigh the risks and consequences of each treatment or nontreatment, and (3) make a choice and communicate that choice (Bramstedt and Arroliga, 2004; Gross and Kazmer, 2006; Scanlon, 2003). In patients who have suspected concomitant brain injury or are receiving mechanical ventilation, it may be difficult if not impossible to determine the patient's decision-making capacity (Scanlon, 2003).

Any request for discontinuation of life-sustaining treatment in a patient with decision-making capacity must be given serious consideration (Beauchamp and Childress, 2001). However, these discussions may pose ethical dilemmas for the staff. Health-care providers may be reluctant to honor such a request early in the recovery period following SCI because multiple studies suggest good quality of life after even high tetraplegia. Also, the severity and irreversibility of the outcome being considered requires exceptional attention to informed consent considerations. If possible, action on the decision should be delayed to give the patient and family time to reconsider the request. In order to assure adequate informed consent for withdrawal of life support, efforts should be made to have patients undergo inpatient rehabilitation, have interactions with persons living in the community with similar injury level and go through a trial of living out of the hospital in order to determine for themselves what quality of life is possible for them. If the patient insists on withdrawal of life support without going through these steps, staff should seek the assistance of an ethics consultant or committee to help them decide whether to act on the request for treatment withdrawal (Bramstedt and Arroliga, 2004). If the patient's choice is clear and unwavering, withdrawal of life-sustaining therapies may be justified ethically. If a patient's request is honored, a mutually agreed-upon plan must be

established to provide not only for the physical comfort of the patient, but also for the emotional and spiritual needs of the patient, family members, and staff. Satisfactory resolution of this dilemma may require involvement of the family or friends, if desired by the patient.

Special Mechanisms of Injury

A small number of nonmechanical injury mechanisms may lead to spinal cord-related patterns of paralysis. Although SCI is most commonly seen following mechanical trauma, it can also be associated with other, less often seen, insults to the cord.

78. Screen for SCI in the patient with high-voltage electrical injury.

(Scientific evidence—II/IV; Grade of recommendation—B; Strength of panel opinion—5)

Rationale: Arevalo et al. (1999) reported 2 cases of injury to the spinal cord among 52 patients admitted with high-voltage electrical injury. The investigators noted that such injuries may occur with or without radiographic or MRI abnormality, leading to a delay in diagnosis. Cherington (1995) described the various types of lightning strikes—direct, side flash, stride (ground) current, and indoor exposure—and the various patterns of manifestation of the injury, including the possibility of SCI from a fall caused by the lightning strike. Ko et al. (2004) noted that features of electrical burn-related spinal cord injury may be transient or may present early or late, and may be progressive, beginning with paraplegia and progressing to tetraplegia. MRI may not be helpful. There is no evidence concerning specific optimal treatment methods. Deficits may improve or may be permanent. Cherington (1995) noted that most patients with SCI from electrical causes are left with a permanent disability.

79. Suspect spinal cord injury in any scuba or commercial diver presenting with neurologic symptoms. Consult with and consider urgent transfer to a hyperbaric unit.

(Scientific evidence—III/IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: Decompression sickness should be strongly considered when divers experience pain or other neurologic symptoms after diving. Barratt and Van Meter (2004) reported that decompression sickness in scuba and commercial divers can present as spinal cord damage, requiring urgent transfer to a recompression chamber if

permanent damage is to be avoided. Neurologic symptoms may be apparent within minutes of ascent from a dive, may be delayed, or may develop during subsequent flight (i.e., ascent to altitude) within a day or so of a dive. MRI findings are variable. Although recompression is ideally achieved within hours, it may be effective up to a few days. Pitkin et al. (1999) reported the prognostic value of the Boussuges scoring system in neurologic decompression illness (Boussuges et al., 1996).

Hysterical Paralysis

Hysterical paralysis is the name given to the condition in which there is partial loss of neurologic function that is inconsistent (i.e., reflexes and loss of power do not match) and/or is not associated with any other evidence of injury, such as abnormality on imaging. It is a diagnosis of exclusion, but it also meets the criteria for a conversion disorder in the DSM-IV-TR (text revision; Letonoff et al., 2002).

80. Consider the diagnosis of hysterical paralysis in patients with marked inconsistencies in neurologic findings.

- Repeat the neurologic exam with great care. Consider using the Spinal Injuries Center test and review base screening imaging, such as plain x-rays.
- Consult in person or by phone with a spinal cord injury specialist before making this diagnosis.
- Encourage the patient gently to resume normal function, minimizing disability.
- Resort to more intensive tests, such as MRI or motor-evoked potential testing, if the patient fails to start improving in 2 to 3 days.

(Scientific evidence—III/IV/V; Grade of recommendation—C; Strength of panel opinion—4)

Rationale: The diagnosis of hysterical paralysis is made if symptoms are inappropriate or out of proportion in comparison to the anatomical or physiologic function and a mental health assessment confirms the presence of psychological factors likely contributing to the onset of paralysis. Reflex function in such cases is nearly always normal.

Apple (1989) reported a series of 17 patients from a single center with various patterns of hysterical paralysis. Twelve of the 17 were male. Two of the patients apparently had reflexes affected by acute alcohol intoxication because they later returned to normal.

The Spinal Injuries Center (SIC) test may assist in this diagnosis (Yugué et al., 2004). In the SIC test, the hips and knees of the supine patient are passively flexed and the feet are on the bed. The test is positive if on withdrawing support the knees stay together. Other investigations may be withheld unless recovery does not begin within a few days (Baker and Silver, 1987). Electrophysiological studies, such as motor-evoked potentials may also help in the diagnosis (Hageman et al., 1993; Jellinek et al., 1992; Letonoff et al., 2002).

If symptoms do not spontaneously remit, the patient may be a candidate for medical rehabilitation with a behaviorally based or strategic-behavioral type of intervention. Mental health referral may also be indicated since somatizing patients frequently have comorbid psychiatric disorders that may influence overall recovery of function.

81. Consider referral to rehabilitation professionals once confident of the hysterical paralysis diagnosis.

(Scientific evidence—IV; Grade of recommendation—C; Strength of panel opinion—5)

Rationale: At least two promising, behaviorally oriented treatments exist for hysterical paralysis. Speed (1996) describes in concrete detail what staff from all rehabilitation disciplines should do to carry out a behavior therapy program that appears encouraging based on a case series. Shapiro and Teasell report an enhancement of the Speed model designed for individuals who may have more chronic symptoms that fail the Speed treatment (Shapiro and Teasell, 2004; Teasell and Shapiro, 1994). Among somatoform patients seen in a general medical setting, other psychiatric disorders, such as major depression, substance abuse, and anxiety disorders, are common comorbid conditions. Successful treatment of these comorbid psychiatric conditions is thought to facilitate overall recovery of function from the conversion disorder (Smith et al., 2000).

Recommendations for Future Research

There are undoubtedly many areas of acute care where further research may help improve our management of the newly-injured person with SCI. The following areas were identified during the evidence review as needing further research:

- Develop a clear and uniform protocol for immobilization and transport of patients with both suspected and proven spinal column and cord injury to minimize further neurological loss and reduce costs to the health-care system.
- Determine optimal endpoints of resuscitation in patients with acute spinal cord injury (SCI) without neurogenic shock.
- Determine evidence-based optimal diagnostic and treatment algorithms for fluid resuscitation, blood product replacement and the use of inotropes and/or vasopressors for neurogenic shock secondary to spinal cord injury.
- Study efficacy of induced moderate hypothermia in acute spinal cord injury.
- The timing of the tertiary trauma survey differs from institution to institution, but typically occurs within 24 hours after admission and is repeated when the patient is awake, responsive, and able to communicate any complaints. The evidence suggests further longitudinal prospective studies are needed to identify optimal timing of the survey.
- Evaluate the design characteristics, necessary protocols for use and effectiveness of pressure distributing mattresses, turning systems and lift devices for prevention of pressure ulcers in the acute phase of SCI.
- Evaluate the optimal diagnostic indications and method of routine screening for blunt cerebrovascular injury in patients with a cervical SCI.
- Determine if decompressive surgery within 8 hours confers any neurologic benefit compared to closed or no reduction. (The Surgical Treatment of Acute Spinal Cord Injury Study is currently ongoing to determine if there is a clinical benefit after early surgical decompression, but a 1999 pilot study showed that it may be technically difficult to achieve surgical decompression within the proposed timeframe.)
- Chronic pain of several types, affecting up to 70% of those with SCI, has been the focus of most recent pain research but relatively little has been written about pain shortly after injury, including whether acute care interventions can affect later pain.
- Further research is needed into the use of optimal mechanical ventilation for acute respiratory failure in SCI with emphasis on mode, rate, and tidal volume, with respect to the first days of care.
- Determine optimal method of weaning for liberation from mechanical ventilation in acute SCI.
- Determine optimal methods for prevention of ventilator-associated pneumonia and other hospital-acquired infections in SCI patients.
- Undertake a prospective randomized controlled trial for prevention of pulmonary embolism in SCI comparing retrievable IVC filters versus anticoagulant methods of prophylaxis after SCI.
- Examine the long-term outcomes of venous thromboembolism after SCI and their impact on recovery and rehabilitation.
- Evaluate the risk of long-term complications of deep vein thrombosis in SCI to determine the relative priority of deep vein thrombosis prevention separate from pulmonary embolism prevention.
- Determine the optimal timing for transition from the indwelling catheter to other methods, such as intermittent catheterization of the bladder.
- Determine nutritional needs—quantity and type—of the newly cord injured human.
- Determine the role, if any, of enrichment with glutamine, considered an option for multiple-trauma patients. Determine the effectiveness of glycemic control on early mortality and morbidity in acute SCI.
- Develop evidence regarding the incremental benefits and risks of each therapeutic physiotherapeutic intervention in the first few days after SCI.

- Define the early psychological sequelae of SCI (e.g., depression, acute stress, grief reactions) that occur during the initial hospitalization for acute SCI and determine the best practices for the diagnosis and management of these conditions. Study the effectiveness of psychosocial interventions for the patient and family, including how to facilitate psychological resilience and how to provide information about prognosis in ways that enhance successful adaptation.
- Describe the prevalence and natural history of requests to withdraw life support.
- Study early interventions (probably open trials) for hysterical paralysis. More natural history studies of hysterical paralysis are needed, for example, to assess the rate and predictors of spontaneous remission as well as the rate of later detection of undiagnosed medical causes.

References

The following list of references includes all sources used by the guideline development panel to support their recommendations. It provides the level of scientific evidence (I–V or NA) for each graded article. A graded article is one that was evaluated by the methodologists to determine whether it met the inclusion criteria established by the panel. If an article is labeled “Scientific Evidence–NA,” it was evaluated by the methodologists but did not meet the level of evidence criteria. If a citation is not labeled, it was not evaluated by the methodologists. Citations labeled NA or unlabeled were included because the panel believes they enhance understanding of the guideline.

- Abd, A. G., and N. M. Braun. Management of life-threatening bradycardia in spinal cord injury. *Chest* 95 (3) (1989): 701–02. **(Scientific evidence–IV)**
- Abel, R., S. Ruf, and B. Spahn. Cervical spinal cord injury and deglutition disorders. *Dysphagia* 19 (2) (2004): 87–94. **(Scientific evidence–IV)**
- Acosta, J. A., J. C. Yang, R. J. Winchell et al. Lethal injuries and time to death in a level I trauma center. *J Am Coll Surg* 186 (5) (1998): 528–33. **(Scientific evidence–IV)**
- Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 342 (18) (2000): 1301–08. **(Scientific evidence–Guideline)**
- Aito, S. Gruppo Italiano Studio Epidemiologico Mielolesioni (GISEM) Group. Complications during the acute phase of traumatic spinal cord lesions. *Spinal Cord* 41 (11) (2003): 629–35. **(Scientific evidence–IV)**
- Aito, S., A. Pieri, M. D’Andrea et al. Primary prevention of deep venous thrombosis and pulmonary embolism in acute spinal cord injured patients. *Spinal Cord* 40 (6) (2002): 300–03. **(Scientific evidence–I)**
- Ajani, A. E., D. J. Cooper, C. D. Scheinkestel et al. Optimal assessment of cervical spine trauma in critically ill patients: A prospective evaluation. *Anaesth Intensive Care* 26 (5) (1998): 487–91. **(Scientific evidence–I)**
- Albert, T. J., M. J. Levine, R. A. Balderston, and J. M. Cotler. Gastrointestinal complications in spinal cord injury. *Spine* 16 (10 Suppl) (1991): S522–25. **(Scientific evidence–III)**
- Albuquerque, F., A. Wolf, C. M. Dunham et al. Frequency of intra-abdominal injury in cases of blunt trauma to the cervical spinal cord. *J Spinal Disord* 5 (4) (1992): 476–80. **(Scientific evidence–II)**
- Allen, C. R., and J. D. Kang. Transient quadriplegia in the athlete. *Clin Sports Med* 21 (1) (2002): 15–27. **(Scientific evidence–V)**
- American Association of Neurological Surgeons and the Congress of Neurological Surgeons. Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons: Guidelines for management of acute cervical spinal injuries. *Neurosurgery* 50 (3 Suppl) (2002): S1. **(Scientific evidence–Guideline)**
- American Psychiatric Association (APA). Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision, *DSM-IV-TR*. **(Scientific evidence–Diagnostic Manual)**
- American Spinal Injury Association. *Guidelines for Facility Categorization and Standards of Care: Spinal Cord Injury*. Atlanta: ASIA, 1981. **(Scientific evidence–Guideline)**
- Annane, D. ICU physicians should abandon the use of etomidate. *Intensive Care Med* 31 (3) (2005): 325–26. **(Scientific Evidence – Guideline)**
- Amin, A., J. Bernard, R. Nadarajah et al. Spinal injuries admitted to a specialist centre over a 5-year period: A study to evaluate delayed admission. *Spinal Cord* 43 (7) (2005): 434–37. **(Scientific evidence–II)**
- Anderson, R. E., B. P. Drayer, B. Braffman et al. Spine trauma. American College of Radiology. ACR Appropriateness Criteria. *Radiology* 215 (2000): 589–95. **(Scientific evidence–V)**
- Apple, D. F., Jr. Hysterical spinal paralysis. *Paraplegia* 27 (6) (1989): 428–31. **(Scientific evidence–IV)**
- Arevalo, J. M., J. A. Lorente, and J. Balseiro-Gomez. Spinal cord injury after electrical trauma treated in a burn unit. *Burns* 25 (5) (1999): 449–52. **(Scientific evidence–II)**
- Attal, N., V. Gaude, L. Brasseur, et al. Intravenous lidocaine in central pain: A double-blind, placebo-controlled, psychophysical study. *Neurology* 54 (3) (2000): 564–74. **(Scientific evidence–I)**
- Attal, N., F. Guirimand, L. Brasseur, et al. Effects of IV morphine in central pain: A randomized placebo-controlled study. *Neurology* 58 (2) (2002): 554–63. **(Scientific evidence–I)**
- Aung, T. S., and W. S. el Masry. Audit of a British centre for spinal injury. *Spinal Cord* 35 (3) (1997): 147–50. **(Scientific evidence–I)**
- Bach, J. R. *Noninvasive Mechanical Ventilation*. Philadelphia: Hanley and Belfus, 2002. **(Scientific evidence–NA)**
- Bach, J. R. Update and perspectives on noninvasive respiratory muscle aids. Part 1: The inspiratory aids. *Chest* 105 (4) (1994a): 1230–40. **(Scientific evidence–V)**
- Bach, J. R. Update and perspectives on noninvasive respiratory muscle aids. Part 2: The expiratory aids. *Chest* 105 (5) (1994b): 1538–44. **(Scientific evidence–V)**
- Bach, J. R., and M. C. Tilton. Life satisfaction and well-being measures in ventilator-assisted individuals with traumatic tetraplegia. *Arch Phys Med Rehabil* 75 (6) (1994): 626–32. **(Scientific evidence–III)**
- Backonja, M., and K. A. Gombar. Response of central pain syndromes to intravenous lidocaine. *Pain Symptom Manage* 7 (1992): 1725–78. **(Scientific evidence–I)**
- Bagnall, A. M., L. Jones, G. Richardson et al. Effectiveness and cost-effectiveness of acute hospital-based spinal cord injuries services: Systematic review. *Health Technol Assess* 7 (19) (2003): iii, 1–92. **(Scientific evidence–Review)**

Baker, J. H., and J. R. Silver. Hysterical paraplegia. *J Neurol Neurosurg Psychiatry* 50 (4) (1987): 375–82. **(Scientific evidence–III)**

Ballard, R. B., G. S. Rozycki, P. G. Newman, et al. An algorithm to reduce the incidence of false-negative FAST examinations in patients at high risk for occult injury: Focused Assessment for the Sonographic Examination of the Trauma patient. *J Am Coll Surg* 189 (2) (1999): 145–50; discussion 150–51. **(Scientific evidence–I)**

Barratt, D. M., and K. Van Meter. Decompression sickness in Miskito Indian lobster divers: Review of 229 cases. *Aviat Space Environ Med* 75 (4) (2004): 350–53. **(Scientific evidence–IV)**

Beauchamp, T. L., and J. F. Childress. *Principles of Biomedical Ethics*. Oxford: Oxford University Press, 2001. **(Scientific evidence–NA)**

Benzel, E. C., B. L. Hart, P. A. Ball et al. Magnetic resonance imaging for the evaluation of patients with occult cervical spine injury. *J Neurosurg* 85 (5) (1996): 824–29. **(Scientific evidence–I)**

Berne, J. D., G. C. Velmahos, Q. El-Tawil et al. Value of complete cervical helical computed tomographic scanning in identifying cervical spine injury in the unevaluable blunt trauma patient with multiple injuries: A prospective study. *J Trauma* 47 (5) (1999): 896–902; discussion 902–3. **(Scientific evidence–I)**

Bilello, J. F., J. W. Davis, M. A. Cunningham et al. Cervical spinal cord injury and the need for cardiovascular intervention. *Arch Surg* 138 (10) (2003): 1127–29. **(Scientific evidence–III)**

Bochicchio, G. V., L. Salzano, M. Joshi et al. Admission preoperative glucose is predictive of morbidity and mortality in trauma patients who require immediate operative intervention. *Am Surg* 71 (2) (2005a): 171–74. **(Scientific evidence–I)**

Bochicchio, G. V., J. Sung, M. Joshi et al. Persistent hyperglycemia is predictive of outcome in critically ill trauma patients. *J Trauma* 58 (5) (2005b): 921–24. **(Scientific evidence–II)**

Boldin, C., J. Raith, F. Fankhauser et al. Predicting neurologic recovery in cervical spinal cord injury with postoperative MR imaging. *Spine* 31 (5) (2006): 554–59. **(Scientific evidence–I)**

Bombardier, C. H., J. S. Richards, J. S. Krause et al. Symptoms of major depression in people with spinal cord injury: Implications for screening. *Arch Phys Med Rehabil* 85 (11) (2004): 1749–56. **(Scientific evidence–IV)**

Bonanno, G. A. Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *Am Psychol* 59 (1) (2004): 20–28. **(Scientific evidence–V)**

Borock, E. C., S. G. Gabram, L. M. Jacobs, and M. A. Murphy. A prospective analysis of a two-year experience using computed tomography as an adjunct for cervical spine clearance. *J Trauma* 31 (7) (1991): 1001–05; discussion 1005–06. **(Scientific evidence–I)**

Boussuges, A., X. Thirion, P. Blanc et al. Neurologic decompression illness: A gravity score. *Undersea Hyperb Med* 23 (3) (1996): 151–55. **(Scientific evidence–III)**

Bracken, M. B., W. F. Collins, D. F. Freeman et al. Efficacy of methylprednisolone in acute spinal cord injury. *JAMA* 251 (1) (1984): 45–52. **(Scientific evidence–I)**

Bracken, M. B., M. J. Shepard, W. F. Collins et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med* 322 (20) (1990): 1405–11. **(Scientific evidence–I)**

Bracken, M. B., M. J. Shepard, T. R. Holford et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. *JAMA* 277 (20) (1997): 1597–1604. **(Scientific evidence–I)**

Bramstedt, K. A., and A. C. Arroliga. On the dilemma of enigmatic refusal of life-saving therapy. *Chest* 126 (2) (2004): 630–33. **(Scientific evidence–V)**

Bravo, G., G. Guizar-Sahagun, A. Ibarra et al. Cardiovascular alterations after spinal cord injury: An overview. *Curr Med Chem Cardiovasc Hematol Agents* 2 (2) (2004): 133–48. **(Scientific evidence–V)**

British Trauma Society. Guidelines for the initial management and assessment of spinal injury. *Injury* 34 (6) (2003): 405–25. **(Scientific evidence–Guideline)**

Brown, L. H., J. E. Gough, and W. B. Simonds. Can EMS providers adequately assess trauma patients for cervical spinal injury? *Prehosp Emerg Care* 2 (1) (1998): 33–36. **(Scientific evidence–IV)**

Brown, M. A., G. Casola, C. B. Sirlin et al. Blunt abdominal trauma: Screening use in 2,693 patients. *Radiology* 218 (2) (2001): 352–58. **(Scientific evidence–I)**

Brown, R. L., M. A. Brunn, and V. F. Garcia. Cervical spine injuries in children: A review of 103 patients treated consecutively at a level 1 pediatric trauma center. *J Pediatr Surg* 36 (8) (2001): 1107–14. **(Scientific evidence–II)**

Buduhan, G., and D. I. McRitchie. Missed injuries in patients with multiple trauma. *J Trauma* 49 (4) (2000): 600–05. **(Scientific evidence–III)**

Burns, A. S., B. S. Lee, J. F. Ditunno Jr., and A. Tessler. Patient selection for clinical trials: The reliability of the early spinal cord injury examination. *J Neurotrauma* 20 (5) (2003): 477–82. **(Scientific evidence–III)**

Burns, S. P., D. G. Golding, W. A. Rolle Jr. et al. Recovery of ambulation in motor-incomplete tetraplegia. *Arch Phys Med Rehabil* 78 (11) (1997): 1169–72. **(Scientific evidence–IV)**

Calancie, B., K. J. Klose, S. Baier, and B. A. Green. Isoflurane-induced attenuation of motor-evoked potentials caused by electrical motor cortex stimulation during surgery. *J Neurosurg* 74 (6) (1991): 897–904. **(Scientific evidence–IV)**

Calancie, B., K. J. Klose, S. Baier, and B. A. Green. Threshold-level multipulse transcranial electrical stimulation of motor cortex for intraoperative monitoring of spinal motor tracts: Description of method and comparison to somatosensory-evoked potential monitoring. *J Neurosurg* 88 (3) (1998): 457–70. **(Scientific evidence–III)**

Calancie, B., M. R. Molano, and J. G. Broton. EMG for assessing the recovery of voluntary movement after acute spinal cord injury in man. *Clin Neurophysiol* 115 (8) (2004): 1748–59. **(Scientific evidence–I)**

Campagnolo, D. I., R. E. Esquieres, and K. J. Kopacz. Effect of timing of stabilization on length of stay and medical complications following spinal cord injury. *J Spinal Cord Med* 20 (3) (1997): 331–34. **(Scientific evidence–III)**

- Castro, F. P., Jr., J. Ricciardi, M. E. Brunet, et al. Stingers, the Torg ratio, and the cervical spine. *Am J Sports Med* 25 (5) (1997): 603–08. **(Scientific evidence–II)**
- Catz, A., A. Zifroni, and O. Philo. Economic assessment of pressure sore prevention using a computerized mattress system in patients with spinal cord injury. *Disabil Rehabil* 27 (21) (2005): 1315–19. **(Scientific evidence–III)**
- Charlifue, S. W., and K. A. Gerhart. Behavioral and demographic predictors of suicide after traumatic spinal cord injury. *Arch Phys Med Rehabil* 72 (7) (1991): 488–92. **(Scientific evidence–III)**
- Chen, Y. and M. J. DeVivo. Epidemiology of extraspinal fractures in acute spinal cord injury: Data from the Model Spinal Cord Injury Care Systems, 1973–1999. *Top Spinal Cord Inj Rehabil* 11 (1) (2005): 18–29. **(Scientific evidence–N/A)**
- Cherington, M. Central nervous system complications of lightning and electrical injuries. *Semin Neurol* 15 (3) (1995): 233–40. **(Scientific evidence–Review)**
- Chesnut, R. M., S. B. Marshall, J. Piek, et al. Early and late systemic hypotension as a frequent and fundamental source of cerebral ischemia following severe brain injury in the Traumatic Coma Data Bank. *Acta Neurochir Suppl* 59 (Wien) (1993): 121–25. **(Scientific evidence–Prognostic, Level I)**
- Chinnock, P., and I. Roberts. Gangliosides for acute spinal cord injury. *Cochrane Database Syst Rev* 2 (2) (2005): CD004444. **(Scientific evidence–Review)**
- Chiu, W. C., B. M. Cushing, A. Rodriguez et al. Abdominal injuries without hemoperitoneum: A potential limitation of focused abdominal sonography for trauma (FAST). *J Trauma* 42 (4) (1997): 617–23; discussion 623–25. **(Scientific evidence–I)**
- Chui, W. H., S. W. Chiu, J. W. Lee, et al. Surgical management of blunt traumatic rupture of the descending thoracic aorta. *Ann Thorac Cardiovasc Surg* 5 (2) (1999): 109–12. **(Scientific evidence–IV)**
- Cline, J. R., E. Scheidel, and E. F. Bigsby. A comparison of methods of cervical immobilization used in patient extrication and transport. *J Trauma* 25 (7) (1985): 649–53. **(Scientific evidence–I)**
- Cocanour, C. S., M. Peninger, B. D. Domonoske, et al. Decreasing ventilator-associated pneumonia in a trauma ICU. *J Trauma* 61 (1) (2006): 122–29; discussion 129–30. **(Scientific evidence–I)**
- Cohan, P., C. Wang, D. L. McArthur, et al. Acute secondary adrenal insufficiency after traumatic brain injury: A prospective study. *Crit Care Med* 33 (10) (2005): 2358–66. **(Scientific evidence–III)**
- Coleman, W. P., D. Benzel, D. W. Cahill, et al. A critical appraisal of the reporting of the National Acute Spinal Cord Injury Studies (II and III) of methylprednisolone in acute spinal cord injury. *J Spinal Disord* 13 (3) (2000): 185–99. **(Scientific evidence–Review)**
- Collier, B., J. Diaz Jr., R. Forbes, et al. The impact of a normoglycemic management protocol on clinical outcomes in the trauma intensive care unit. *JPEN J Parenter Enteral Nutr* 29 (5) (2005): 353–58; discussion 359. **(Scientific evidence–I)**
- Connell, R. A., C. A. Graham, and P. T. Munro. Is spinal immobilisation necessary for all patients sustaining isolated penetrating trauma? *Injury* 34 (12) (2003): 912–14. **(Scientific evidence–IV)**
- Conrad, S. A., A. Gabrielli, B. Margolis, et al. Randomized, double-blind comparison of immediate-release omeprazole oral suspension versus intravenous cimetidine for the prevention of upper gastrointestinal bleeding in critically ill patients. *Crit Care Med* 33 (4) (2005): 760–65. **(Scientific evidence–I)**
- Consortium for Spinal Cord Medicine. *Depression Following Spinal Cord Injury: A Clinical Practice Guideline for Primary Care Physicians*. Washington, DC: Paralyzed Veterans of America, 1998. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. Clinical practice guidelines: Neurogenic bowel management in adults with spinal cord injury. *J Spinal Cord Med* 21 (3) (1998): 248–93. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. *Clinical Practice Guidelines: Neurogenic Bowel Management in Adults with Spinal Cord Injury*. Washington, DC: Paralyzed Veterans of America, 1998. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. *Outcomes Following Traumatic Spinal Cord Injury: Clinical Practice Guidelines for Health-Care Professionals*. Washington, DC: Paralyzed Veterans of America, 1999. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. *Prevention of Thromboembolism in Spinal Cord Injury*, 2nd edition. Washington, DC: Paralyzed Veterans of America, 1999. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals*. Washington, DC: Paralyzed Veterans of America, 2000. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. *Respiratory Management Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals*. Washington, DC: Paralyzed Veterans of America, 2005. **(Scientific evidence–Guideline)**
- Consortium for Spinal Cord Medicine. *Bladder Management for Adults with Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Providers*. Washington, DC: Paralyzed Veterans of America, 2006. **(Scientific evidence–Guideline)**
- Cook, D., G. Guyatt, J. Marshall, et al. A comparison of sucralfate and ranitidine for the prevention of upper gastrointestinal bleeding in patients requiring mechanical ventilation. Canadian Critical Care Trials Group. *N Engl J Med* 338 (12) (1998): 791–97. **(Scientific evidence–I)**
- Cook, D., D. Heyland, L. Griffith, et al. Risk factors for clinically important upper gastrointestinal bleeding in patients requiring mechanical ventilation. Canadian Critical Care Trials Group. *Crit Care Med* 27 (12) (1999): 2812–17. **(Scientific evidence–I)**
- Cook, D. J., H. D. Fuller, G. H. Guyatt, et al. Risk factors for gastrointestinal bleeding in critically ill patients. Canadian Critical Care Trials Group. *N Engl J Med* 330 (6) (1994): 377–81. **(Scientific evidence–IV)**
- Cooper, C., L. Carbone, C. J. Michet, et al. Fracture risk in patients with ankylosing spondylitis: A population-based study. *J Rheumatol* 21 (10) (1994): 1877–82. **(Scientific evidence–III)**

- Cornwell, E. E., III, D. C. Chang, J. P. Bonar, et al. Thoracolumbar immobilization for trauma patients with torso gunshot wounds: Is it necessary? *Arch Surg* 136 (3) (2001): 324–27. **(Scientific evidence–IV)**
- Costa, P., Bruno, A., Bonzanino, M., et al. Somatosensory- and motor-evoked potential monitoring during spine and spinal cord surgery. *Spinal Cord* 45 (1): 86–91 (2007). Epub 2006 May 2.
- Cothren, C. C., and E. E. Moore. Blunt cerebrovascular injuries. *Clinics* 60 (6) (2005): 489–96. **(Scientific evidence–Review)**
- Cothren, C. C., E. E. Moore, W. L. Biffl, et al. Cervical spine fracture patterns predictive of blunt vertebral artery injury. *J Trauma* 55 (5) (2003): 811–13. **(Scientific evidence–IV)**
- Cotton, B. A., J. P. Pryor, I. Chinwalla, et al. Respiratory complications and mortality risk associated with thoracic spine injury. *J Trauma* 59 (6) (2005): 1400–07; discussion 1407–09. **(Scientific evidence–II)**
- Cox, S. A., S. M. Weiss, E. A. Posuniak, et al. Energy expenditure after spinal cord injury: An evaluation of stable rehabilitating patients. *J Trauma* 25 (5) (1985): 419–23. **(Scientific evidence–IV)**
- Curt, A., and V. Dietz. Ambulatory capacity in spinal cord injury: Significance of somatosensory-evoked potentials and ASIA protocol in predicting outcome. *Arch Phys Med Rehabil* 78 (1) (1997): 39–43. **(Scientific evidence–I)**
- Dalyan, M., A. Sherman, and D. D. Cardenas. Factors associated with contractures in acute spinal cord injury. *Spinal Cord* 36 (6) (1998): 405–08. **(Scientific evidence–II)**
- Darsaut, T. E., R. Ashforth, R. Bhargava, et al. A pilot study of magnetic resonance imaging-guided closed reduction of cervical spine fractures. *Spine* 31 (18) (2006): 2085–90. **(Scientific evidence–IV)**
- Davidoff, G., R. Doljanac, S. Berent, et al. Galveston Orientation and Amnesia Test: Its utility in the determination of closed head injury in acute spinal cord injury patients. *Arch Phys Med Rehabil* 69 (6) (1988a): 432–34. **(Scientific evidence–II)**
- Davidoff, G., J. Morris, E. Roth, and J. Bleiberg. Closed head injury in spinal cord injured patients: Retrospective study of loss of consciousness and posttraumatic amnesia. *Arch Phys Med Rehabil* 66 (1) (1985a): 41–43. **(Scientific evidence–Review)**
- Davidoff, G., J. Morris, E. Roth, and J. Bleiberg. Cognitive dysfunction and mild closed head injury in traumatic spinal cord injury. *Arch Phys Med Rehabil* 66 (8) (1985b): 489–91. **(Scientific evidence–II)**
- Davidoff, G., P. Thomas, M. Johnson, et al. Closed head injury in acute traumatic spinal cord injury: Incidence and risk factors. *Arch Phys Med Rehabil* 69 (10) (1988b): 869–72. **(Scientific evidence–II)**
- Davies, G., C. Deakin, and A. Wilson. The effect of a rigid collar on intracranial pressure. *Injury* 27 (9) (1996): 647–49. **(Scientific evidence–IV)**
- Davis, J. W., D. L. Phreaner, D. B. Hoyt, and R. C. Mackerseie. The etiology of missed cervical spine injuries. *J Trauma* 34 (3) (1993): 342–46. **(Scientific evidence–II)**
- De Lorenzo, R. A. A review of spinal immobilization techniques. *J Emerg Med* 14 (5) (1996): 603–13. **(Scientific evidence–Review)**
- Deep, K., M. V. Jigajinni, A. N. McLean, and M. H. Fraser. Prophylaxis of thromboembolism in spinal injuries: Results of enoxaparin used in 276 patients. *Spinal Cord* 39 (2) (2001): 88–91. **(Scientific evidence–IV)**
- Demetriades, D., M. Martin, A. Salim, et al. The effect of trauma center designation and trauma volume on outcome in specific severe injuries. *Ann Surg* 242 (4) (2005): 512–17; discussion 517–19. **(Scientific evidence–III)**
- Demetriades, D., M. Martin, A. Salim, et al. Relationship between American College of Surgeons trauma center designation and mortality in patients with severe trauma (injury severity score > 15). *J Am Coll Surg* 202 (2) (2006): 212–15; quiz A45. **(Scientific evidence–III)**
- DeVivo, M. J., P. L. Kartus, S. L. Stover, and P. R. Fine. Benefits of early admission to an organised spinal cord injury care system. *Paraplegia* 28 (9) (1990): 545–55. **(Scientific evidence–III)**
- Dewar, A. Nurses' experiences in giving bad news to patients with spinal cord injuries. *J Neurosci Nurs* 32 (6) (2000): 324–30. **(Scientific evidence–IV)**
- Dial, S., J. A. Delaney, A. N. Barkun, and S. Suissa. Use of gastric acid-suppressive agents and the risk of community-acquired *Clostridium difficile*-associated disease. *JAMA* 294 (23) (2005): 2989–95. **(Scientific evidence–III)**
- Dixit, S. Bradycardia associated with high cervical spinal cord injury. *Surg Neurol* 43 (5) (1995): 514. **(Scientific evidence–NA)**
- Dodek, P., S. Keenan, D. Cook, et al. Canadian Critical Care Trials Group and Canadian Critical Care Society. Evidence-based clinical practice guideline for the prevention of ventilator-associated pneumonia. *Ann Intern Med* 141 (4) (2004): 305–13. **(Scientific evidence–Guideline)**
- Domeier, R. M. Indications for prehospital spinal immobilization. National Association of EMS Physicians Standards and Clinical Practice Committee. *Prehosp Emerg Care* 3 (3) (1999): 251–53. **(Scientific evidence–Guideline)**
- Domeier, R. M., R. W. Evans, R. A. Swor, et al. The reliability of prehospital clinical evaluation for potential spinal injury is not affected by the mechanism of injury. *Prehosp Emerg Care* 3 (4) (1999): 332–37. **(Scientific evidence–I)**
- Drakulovic, M. B., A. Torres, T. T. Bauer, et al. Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: A randomised trial. *Lancet* 354 (9193) (1999): 1851–58. **(Scientific evidence–I)**
- Dries, D. J., M. D. McGonigal, M. S. Malian, et al. Protocol-driven ventilator weaning reduces use of mechanical ventilation, rate of early reintubation, and ventilator-associated pneumonia. *J Trauma* 56 (5) (2004): 943–51; discussion 951–52. **(Scientific evidence–IV)**
- Duh, M. S., M. J. Shepard, J. E. Wilberger, and M. B. Bracken. The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. *Neurosurgery* 35 (2) (1994): 240–48; discussion 248–49. **(Scientific evidence–III)**
- Duperier, T., A. Mosenthal, K. G. Swan, and S. Kaul. Acute complications associated with greenfield filter insertion in high-risk trauma patients. *J Trauma* 54 (3) (2003): 545–49. **(Scientific evidence–IV)**

- Dvorak, M. F., V. K. Noonan, L. Belanger, et al. Early versus late enteral feeding in patients with acute cervical spinal cord injury: A pilot study. *Spine* 29 (9) (2004): E175–80. **(Scientific evidence–I)**
- Eide, P. K., A. Stubhaug, A. E. Stenehjem. Central dysesthesia pain after traumatic spinal cord injury is dependent on N-methyl-D-aspartate receptor activation. *J Neurosurgery* 37 (6) (1995): 1080–87. **(Scientific evidence–I)**
- Eastman, A. L., D. P. Chason, C. L. Perez, et al. Computed tomographic angiography for the diagnosis of blunt cervical vascular injury: Is it ready for primetime? *J Trauma* 60 (5) (2006): 925–29; discussion 929. **(Scientific evidence–I)**
- Elliott, T. R., and R. G. Frank. Depression following spinal cord injury. *Arch Phys Med Rehabil* 77 (8) (1996): 816–23. **(Scientific evidence–Review)**
- Enderson, B. L., D. B. Reath, J. Meadors, et al. The tertiary trauma survey: A prospective study of missed injury. *J Trauma* 30 (6) (1990): 666–69; discussion 669–70. **(Scientific evidence–IV)**
- Evans, E., N. Turley, N. Robinson, and M. Clancy. Randomised controlled trial of patient-controlled analgesia compared with nurse-delivered analgesia in an emergency department. *Emerg Med J* 22 (1) (2005): 25–29. **(Scientific evidence–I)**
- Farahmand, N., C. B. Sirlin, M. A. Brown et al. Hypotensive patients with blunt abdominal trauma: Performance of screening US. *Radiology* 235 (2) (2005): 436–43. **(Scientific evidence–I)**
- Fehlings, M. G., and R. G. Perrin. The role and timing of early decompression for cervical spinal cord injury: Update with a review of recent clinical evidence. *Injury* 36 (2) (2005): B13–26. **(Scientific evidence–Review)**
- Feldtkeller, E., D. Vosse, P. Geusens, and S. Van der Linden. Prevalence and annual incidence of vertebral fractures in patients with ankylosing spondylitis. *Rheumatol* 26 (3) (2006): 234–39. **(Scientific evidence–NA)**
- Finnerup N. B., F. Biering-Sorensen, I. L. Johannesen et al. Intravenous lidocaine relieves spinal cord injury pain: a randomized controlled trial. *Anesthesiology* (2005): 102: 1023–30. **(Scientific evidence–I)**
- Fishburn, M. J., R. J. Marino, and J. F. Ditunno Jr. Atelectasis and pneumonia in acute spinal cord injury. *Arch Phys Med Rehabil* 71 (3) (1990): 197–200. **(Scientific evidence–IV)**
- Flanders, A. E., C. M. Spettell, L. M. Tartaglino et al. Forecasting motor recovery after cervical spinal cord injury: Value of MR imaging. *Radiology* 201 (3) (1996): 649–55. **(Scientific evidence–I)**
- Franga, D. L., M. L. Hawkins, R. S. Medeiros, and D. Adewumi. Recurrent asystole resulting from high cervical spinal cord injuries. *Am Surg* 72 (6) (2006): 525–29. **(Scientific evidence–IV)**
- Frempong-Boadu, A., J. K. Houten, B. Osborn et al. Swallowing and speech dysfunction in patients undergoing anterior cervical discectomy and fusion: A prospective, objective preoperative and postoperative assessment. *J Spinal Disord* 15 (5) (2002): 362–68. **(Scientific evidence–II)**
- Friedman, D., A. Flanders, C. Thomas, and W. Millar. Vertebral artery injury after acute cervical spine trauma: Rate of occurrence as detected by MR angiography and assessment of clinical consequences. *ARJ Am J Roentgenol* 164 (2) (1995): 443–47; discussion 448–49. **(Scientific evidence–I)**
- Fullerton, D. T., R. F. Harvey, M. H. Klein, and T. Howell. Psychiatric disorders in patients with spinal cord injuries. *Arch Gen Psychiatry* 38 (12) (1981): 1369–71. **(Scientific evidence–IV)**
- Garber, A. J., E. S. Moghissi, E. D. Bransome Jr., et al. American College of Endocrinology Task Force on Inpatient Diabetes Metabolic Control. American College of Endocrinology position statement on inpatient diabetes and metabolic control. *Endocr Pract* 10 (1) (2004): 77–82. **(Scientific evidence–NA)**
- Garcia-de-Lorenzo, A., A. Zarazaga, P. P. Garcia-Luna, et al. Clinical evidence for enteral nutritional support with glutamine: A systematic review. *Nutrition* 37 (6) (2003): 805–11. **(Scientific evidence–Review)**
- Garcia-Zozaya, I. A. Adrenal insufficiency in acute spinal cord injury. *J Spinal Cord Med* 29 (1) (2006): 67–69. **(Scientific evidence–IV)**
- Garland, D. E., T. V. Rieser, and D. I. Singer. Treatment of femoral shaft fractures associated with acute spinal cord injuries. *Clin Orthop Relat Res* (197) (1985): 191–95. **(Scientific evidence–III)**
- Garland, D. E., T. Saucedo, and T. V. Reiser. The management of tibial fractures in acute spinal cord injury patients. *Clin Orthop Relat Res* (213) (1986): 237–40. **(Scientific evidence–IV)**
- Garstang, S. V., S. C. Kirshblum, and K. E. Wood. Patient preference for in-exsufflation for secretion management with spinal cord injury. *J Spinal Cord Med* 23 (2) (2000): 80–85. **(Scientific evidence–IV)**
- Geerts, W. H., G. F. Pineo, J. A. Heit et al. Prevention of venous thromboembolism: The seventh ACCP conference on antithrombotic and thrombolytic therapy. *Chest* 126 (3 Suppl) (2004): 338S–400S. **(Scientific evidence–Review)**
- Gerhart, K. A., J. Koziol-McLain, S. R. Lowenstein, and G. G. Whiteneck. Quality of life following spinal cord injury: Knowledge and attitudes of emergency care providers. *Ann Emerg Med* 23 (4) (1994): 807–12. **(Scientific evidence–NA)**
- Goetz, L. L., G. S. Brown, and M. M. Priebe. Interface pressure characteristics of alternating air cell mattresses in persons with spinal cord injury. *J Spinal Cord Med* 25 (3) (2002): 167–73. **(Scientific evidence–III)**
- Gondim, F. A., A. C. Lopes Jr., G. R. Oliveira et al. Cardiovascular control after spinal cord injury. *Curr Vasc Pharmacol* 2 (1) (2004): 71–79. **(Scientific evidence–Review)**
- Gordon, S. A., K. H. Stage, K. E. Tansey, and Y. Lotan. Conservative management of priapism in acute spinal cord injury. *Urology* 65 (6) (2005): 1195–97. **(Scientific evidence–II)**
- Gramlich, L., K. Kichian, J. Pinilla, et al. Does enteral nutrition compared to parenteral nutrition result in better outcomes in critically ill adult patients? A systematic review of the literature. *Nutrition* 20 (10) (2004): 843–48. **(Scientific evidence–Review)**
- Griffiths, J., V. S. Barber, L. Morgan, and J. D. Young. Systematic review and meta-analysis of studies of the timing of tracheostomy in adult patients undergoing artificial ventilation. *BMJ* 330 (7502) (2005): 1243. **(Scientific evidence–Review)**
- Gronert, G. A. and R. A. Theye. Pathophysiology of hyperkalemia induced by succinylcholine. *Anesthesiology* 43 (1) (1975): 89–99. **(Scientific evidence–V)**

- Gross, J., and J. Kazmer. Depression and the MDS. *Provider* 32 (5) (2006): 33–36. **(Scientific evidence–NA)**
- Gunby, I. New focus on spinal cord injury. *JAMA* 245 (12) (1981): 1201–06. **(Scientific evidence–Guideline)**
- Gupta, A., and W. S. el Masri. Multilevel spinal injuries: incidence, distribution, and neurological patterns. *J Bone Joint Surg Br* 71 (4) (1989): 692–95. **(Scientific evidence–IV)**
- Hadley, J. S. and C. J. Hinds. Anabolic strategies in critical illness. *Curr Opin Pharmacol* 2 (6) (2002): 700–07. **(Scientific evidence–Review)**
- Hageman, G., C. J. Eertman-Meyer, and J. Tel-Hampsink. The clinical diagnostic value of studies using magnetic stimulation. *Ned Tijdschr Geneeskde* 137 (45) (1993): 2323–28. **(Scientific evidence–IV)**
- Hall, K. M., S. T. Knudsen, J. Wright et al. Follow-up study of individuals with high tetraplegia (C1–C4) 14 to 24 years postinjury. *Arch Phys Med Rehabil* 80 (11) (1999): 1507–13. **(Scientific evidence–IV)**
- Harrop, J. S., A. D. Sharan, E. H. Scheid Jr. et al. Tracheostomy placement in patients with complete cervical spinal cord injuries: American Spinal Injury Association Grade A. *J Neurosurg* 100 (1 Suppl Spine) (2004): 20–23. **(Scientific evidence–IV)**
- Hauswald, M., G. Tandberg, Z. Omar. Out-of-hospital spinal immobilization: Its effect on neurologic injury. *Acad Emerg Med* 5 (3) (1998): 214–19. **(Scientific evidence–III)**
- Heary, R. F., A. R. Vaccaro, J. J. Mesa, and R. A. Balderston. Thoracolumbar infections in penetrating injuries to the spine. *Orthop Clin North Am* 27 (1) (1996): 69–81. **(Scientific evidence–Review)**
- Heenan, C. M., and U. Piotrowski. Creation of a client goal-setting framework. *SCI Nurs* 17 (4) (2000): 153–61. **(Scientific evidence–NA)**
- Heinemann, A. W., G. M. Yarkony, E. J. Roth, et al. Functional outcome following spinal cord injury: A comparison of specialized spinal cord injury center vs general hospital short-term care. *Arch Neurol* 46 (10) (1989): 1098–102. **(Scientific evidence–III)**
- Helling, T. S., M. Watkins, L. L. Evans, et al. Low falls: An underappreciated mechanism of injury. *J Trauma* 46 (3) (1999): 453–56. **(Scientific evidence–II)**
- Heyland, D. K., R. Dhaliwal, J. W. Drover, et al. Canadian Critical Care Clinical Practice Guidelines Committee. Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. *JPEN J Parenter Enteral Nutr* 27 (5) (2003): 355–73. **(Scientific evidence–Review)**
- Hitchon, P. W., A. M. From, M. D. Brenton, et al. Fractures of the thoracolumbar spine complicating ankylosing spondylitis. *J Neurosurg* 97 (2 Suppl) (2002): 218–22. **(Scientific evidence–IV)**
- Holly, L. T., D. F. Kelly, G. J. Counelis, et al. Cervical spine trauma associated with moderate and severe head injury: Incidence, risk factors, and injury characteristics. *J Neurosurg* 96 (3 Suppl) (2002): 285–91. **(Scientific evidence–II)**
- Hurlbert, R. J. Methylprednisolone for acute spinal cord injury: An inappropriate standard of care. *J Neurosurg* 93 (1 Suppl) (2000): 1–7. **(Scientific evidence–Review)**
- Hurlbert, R. J. Strategies of medical intervention in the management of acute spinal cord injury. *Spine* 31 (11 Suppl) (2006): S16–21; discussion S36. **(Scientific evidence–Review)**
- Iida, H., S. Tachibana, T. Kitahara, et al. Association of head trauma with cervical spine injury, spinal cord injury, or both. *J Trauma* 46 (3) (1999): 450–52. **(Scientific evidence–II)**
- Inaba, K., A. W. Kirkpatrick, J. Finkelstein, et al. Blunt abdominal aortic trauma in association with thoracolumbar spine fractures. *Injury* 32 (3) (2001): 201–07. **(Scientific evidence–IV)**
- Jackson, A. B., and T. E. Groomes. Incidence of respiratory complications following spinal cord injury. *Arch Phys Med Rehabil* 75 (3) (1994): 270–75. **(Scientific evidence–IV)**
- Jacobi, J., G. L. Fraser, D. B. Coursin, et al. Task Force of the American Co. Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult. *Crit Care Med* 30 (1) (2002): 119–41. **(Scientific evidence–Guideline)**
- Jacobs, S. R., N. K. Yeane, G. J. Herbison, and J. F. Ditunno, Jr. Future ambulation prognosis as predicted by somatosensory-evoked potentials in motor complete and incomplete quadriplegia. *Arch Phys Med Rehabil* 76 (7) (1995): 635–41. **(Scientific evidence–IV)**
- Jastrzab, G., and K. E. Khor. Use of breath-activated patient-controlled analgesia for acute pain management in a patient with quadriplegia. *Spinal Cord* 37 (3) (1999): 221–23. **(Scientific evidence–IV)**
- Jellinek, D. A., R. Bradford, I. Bailey, and L. Symon. The role of motor-evoked potentials in the management of hysterical paraplegia: case report. *Paraplegia* 30 (4) (1992): 300–02. **(Scientific evidence–IV)**
- Jeremitsky, E., L. A. Omert, C. M. Dunham, et al. The impact of hyperglycemia on patients with severe brain injury. *J Trauma* 58 (1) (2005): 47–50. **(Scientific evidence–IV)**
- Johnson, J. R., M. A. Kuskowski, and T. J. Wilt. Systematic review: Antimicrobial urinary catheters to prevent catheter-associated urinary tract infection in hospitalized patients. *Ann Intern Med* 144 (2) (2006): 116–26. **(Scientific evidence–Review)**
- Jones, L., and A. Bagnall. Spinal injuries centres (SICs) for acute traumatic spinal cord injury. *Cochrane Database Syst Rev* 4 (4) (2004): CD004442. **(Scientific evidence–Review)**
- Jones, T., V. Ugalde, P. Franks, et al. Venous thromboembolism after spinal cord injury: Incidence, time course, and associated risk factors in 16,240 adults and children. *Arch Phys Med Rehabil* 86 (12) (2005): 2240–47. **(Scientific evidence–IV)**
- Judd, F. K., and D. J. Brown. Psychiatry in the spinal injuries unit. *Paraplegia* 25 (3) (1987): 254–57. **(Scientific evidence–NA)**
- Judd, F. K., J. Stone, J. E. Webber, et al. Depression following spinal cord injury: A prospective inpatient study. *Br J Psychiatry* 154 (1989): 668–71. **(Scientific evidence–IV)**
- Kennedy, P., J. Duff, M. Evans, and A. Beedie. Coping Effectiveness Training reduces depression and anxiety following traumatic spinal cord injuries. *Brit J Clin Psych* 42 (Pt 1) (2003): 41–52. **(Scientific evidence–NA)**
- Khetarpal, S., B. S. Steinbrunn, M. D. McGonigal, et al. Trauma faculty and trauma team activation: Impact on trauma system function and patient outcome. *J Trauma* 47 (3) (1999): 576–81. **(Scientific evidence–III)**
- Kihtiir, T., R. R. Ivatury, R. Simon, and W. M. Stahl. Management of transperitoneal gunshot wounds of the spine. *J Trauma* 31 (12) (1991): 1579–83. **(Scientific evidence–IV)**

- Kirshblum, S., M. V. Johnston, J. Brown, et al. Predictors of dysphagia after spinal cord injury. *Arch Phys Med Rehabil* 80 (9) (1999): 1101–05. **(Scientific evidence–III)**
- Kirshblum, S. C., and K. C. O'Connor. Predicting neurologic recovery in traumatic cervical spinal cord injury. *Arch Phys Med Rehabil* 79 (11) (1998): 1456–66. **(Scientific evidence–Review)**
- Kishi, Y., R. G. Robinson, and J. T. Kosier. Suicidal ideation among patients with acute life-threatening physical illness: Patients with stroke, traumatic brain injury, myocardial infarction, and spinal cord injury. *Psychosomatics* 42 (5) (2001): 382–90. **(Scientific evidence–I)**
- Kiwerski, J. Bleeding from the alimentary canal during the management of spinal cord injury patients. *Paraplegia* 24 (2) (1986): 92–96. **(Scientific evidence–IV)**
- Klein, Y., S. M. Cohn, D. Soffer et al. Spine injuries are common among asymptomatic patients after gunshot wounds. *J Trauma* 58 (4) (2005): 833–36. **(Scientific evidence–II)**
- Ko, S. H., W. Chun, and H. C. Kim. Delayed spinal cord injury following electrical burns: A 7-year experience. *Burns* 30 (7) (2004): 691–95. **(Scientific evidence–IV)**
- Koeman, M., A. J. A. M. van der Ven, E. Hak, et al. Oral decontamination with Chlorhexidine reduces the incidence of ventilator-associated pneumonia. *Am J Respir Crit Care Med* 173 (12) (2006): 1348–55. **(Scientific evidence–I)**
- Kraft, M. R. Refusal of treatment: An ethical dilemma. *SCI Nurs* 16 (1) (1999): 9–13, 20. **(Scientific evidence–NA)**
- Krassioukov, A., and V. E. Claydon. The clinical problems in cardiovascular control following spinal cord injury: An overview. *Prog Brain Res* 152 (2006): 223–29. **(Scientific evidence–Review)**
- Krause, J. S., B. Kemp, and J. Coker. Depression after spinal cord injury: Relation to gender, ethnicity, aging, and socioeconomic indicators. *Arch Phys Med Rehabil* 81 (8) (2000): 1099–109. **(Scientific evidence–I)**
- Kress, J. P., A. S. Pohlman, M. F. O'Connor, and J. B. Hall. Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation. *N Engl J Med* 342 (20) (2000): 1471–77. **(Scientific evidence–I)**
- Kübler-Ross, E. *On Death and Dying*. London: Tavistock Publications, 1969. **(Scientific evidence–NA)**
- Kuric, J., C. E. Lucas, A. M. Ledgerwood, et al. Nutritional support: A prophylaxis against stress bleeding after spinal cord injury. *Paraplegia* 27 (2) (1989): 140–45. **(Scientific evidence–III)**
- Kvarnstrom, A., R. Karlsten, H. Quiding, T. Gordh. The analgesic effect of intravenous ketamine and lidocaine on pain after spinal cord injury. *Acta Anaesthesiologica Scandinavica* 48 (2004): 498–506. **(Scientific evidence–I)**
- La Rosa, G., A. Conti, S. Cardali, et al. Does early decompression improve neurological outcome of spinal cord injured patients? Appraisal of the literature using a meta-analytical approach. *Spinal Cord* 42 (9) (2004): 503–12. **(Scientific evidence–Review)**
- Laskowski, S., and J. Morse. The patient with spinal cord injury: The modification of hope and expressions of despair. *Can J of Rehabil* 6 (1993): 143–53. **(Scientific evidence–Guideline)**
- Leeman, C. P. Depression and the right to die. *Gen Hosp Psychiatry* 21 (2) (1999): 112–15. **(Scientific evidence–NA)**
- Lehmann, K. G., J. G. Lane, J. M. Piepmeier, and W. P. Batsford. Cardiovascular abnormalities accompanying acute spinal cord injury in humans: Incidence, time course, and severity. *J Am Coll Cardiol* 10 (1) (1987): 46–52. **(Scientific evidence–IV)**
- Letonoff, E. J., T. R. Williams, and K. S. Sidhu. Hysterical paralysis: A report of three cases and a review of the literature. *Spine* 27 (20) (2002): E441–45. **(Scientific evidence–Review)**
- Levendoglu, F., C. O. Oğün, O. Ozerbil, T. C. Oğün and H. Ugurlu. Gabapentin is a first line drug for the treatment of neuropathic pain in spinal cord injury. *Spine* 29 (7) (2004): 743–51. **(Scientific evidence–I)**
- Levi, L., A. Wolf, and H. Belzberg. Hemodynamic parameters in patients with acute cervical cord trauma: Description, intervention, and prediction of outcome. *Neurosurgery* 33 (6) (1993): 1007–16; discussion 1016–17. **(Scientific evidence–II)**
- Lifshutz, J., Z. Lidar, and D. Maiman. Thoracic aortic pseudoaneurysm after spine trauma in ankylosing spondylitis. Case report. *J Neurosurg Spine* 2 (2) (2005): 218–21. **(Scientific evidence–IV)**
- Linares, H. A., A. R. Mawson, E. Suarez, and J. J. Biundo. Association between pressure sores and immobilization in the immediate postinjury period. *Orthopedics* 10 (4) (1987): 571–73. **(Scientific evidence–II)**
- Lohne, V., and E. Severinsson. Hope and despair: The awakening of hope following acute spinal cord injury—an interpretative study. *Int J Nurs Stud* 41 (8) (2004a): 881–90. **(Scientific evidence–IV)**
- Lohne, V., and E. Severinsson. Hope during the first months after acute spinal cord injury. *J Adv Nurs* 47 (3) (2004b): 279–86. **(Scientific evidence–IV)**
- Lohne, V., and E. Severinsson. Patients' experiences of hope and suffering during the first year following acute spinal cord injury. *J Clin Nurs* 14 (3) (2005): 285–93. **(Scientific evidence–IV)**
- Lynch, M. E., A. J. Clark and J. Sawynok. A pilot study examining topical amitriptyline, ketamine, and a combination of both in the treatment of neuropathic pain. *Clin J Pain* 19 (2003): 323–28. **(Scientific evidence–I)**
- Macdonell, R. A., and G. A. Donnan. Magnetic cortical stimulation in acute spinal cord injury. *Neurology* 45 (2) (1995): 303–06. **(Scientific evidence–III)**
- MacKenzie, E. J., F. P. Rivara, G. J. Jurkovich, et al. A national evaluation of the effect of trauma-center care on mortality. *N Engl J Med* 354 (4) (2006): 366–78. **(Scientific evidence–III)**
- Marino, R. J., J. F. Ditunno Jr., W. H. Donovan, and F. Maynard Jr. Neurologic recovery after traumatic spinal cord injury: Data from the Model Spinal Cord Injury Systems. *Arch Phys Med Rehabil* 80 (11) (1999): 1391–96. **(Scientific evidence–IV)**
- Marino, R. J. *International standards for neurological classification of spinal cord injury*. Atlanta: American Spinal Cord Injury Association, 2002. **(Scientific evidence–NA)**
- Marshall, L. F., S. Knowlton, S. R. Garfin, et al. Deterioration following spinal cord injury: A multicenter study. *J Neurosurg* 66 (3) (1987): 400–04. **(Scientific evidence–IV)**
- Martyn, J. A. and M. Richtsfeld. Succinylcholine-induced hyperkalemia in acquired pathologic states: Etiologic factors and molecular mechanisms. *Anesthesiology* 104 (1) (2006): 158–69. **(Scientific evidence–Report)**

- Martz, E., H. Livneh, M. Priebe, et al. Predictors of psychosocial adaptation among people with spinal cord injury or disorder. *Arch Phys Med Rehabil* 86 (6) (2005): 1182–92. **(Scientific evidence–NA)**
- Mawson, A. R., J. J. Biundo Jr., P. Neville, et al. Risk factors for early occurring pressure ulcers following spinal cord injury. *Am J Phys Med Rehabil* 67 (3) (1988): 123–27. **(Scientific evidence–IV)**
- Maxwell, R. A., M. Chavarria-Aguilar, W. T. Cockerham, et al. Routine prophylactic vena cava filtration is not indicated after acute spinal cord injury. *J Trauma* 52 (5) (2002): 902–06. **(Scientific evidence–III)**
- McCulloch, P. T., J. France, D. L. Jones, et al. Helical computed tomography alone compared with plain radiographs with adjunct computed tomography to evaluate the cervical spine after high-energy trauma. *J Bone Joint Surg Am* 87 (11) (2005): 2388–94. **(Scientific evidence–I)**
- Medzon, R., T. Rothenhaus, C. M. Bono, et al. Stability of cervical spine fractures after gunshot wounds to the head and neck. *Spine* 30 (20) (2005): 2274–79. **(Scientific evidence–IV)**
- Miller, M. T., M. D. Pasquale, W. J. Bromberg, et al. Not so FAST. *J Trauma* 54 (1) (2003): 59–60. **(Scientific evidence–I)**
- Miller, P. R., T. C. Fabian, M. A. Croce, et al. Prospective screening for blunt cerebrovascular injuries: Analysis of diagnostic modalities and outcomes. *Ann Surg* 236 (3) (2002): 386–93; discussion 393–95. **(Scientific evidence–I)**
- Mirovsky, Y., E. Shalmon, A. Blankstein, and N. Halperin. Complete paraplegia following gunshot injury without direct trauma to the cord. *Spine* 30 (21) (2005): 2436–38. **(Scientific evidence–II)**
- Mitra, D., D. M. Elvins, D. J. Speden, and A. J. Collins. The prevalence of vertebral fractures in mild ankylosing spondylitis and their relationship to bone mineral density. *Rheumatol* 39 (1) (2000): 85–89. **(Scientific evidence–III)**
- Mobbs, R. J., M. A. Stoodley, and J. Fuller. Effect of cervical hard collar on intracranial pressure after head injury. *ANZ J Surg* 72 (6) (2002): 389–91. **(Scientific evidence–III)**
- Morishima, N., K. Ohta, and Y. Miura. The influences of halo-vest fixation and cervical hyperextension on swallowing in healthy volunteers. *Spine* 30 (7) (2005): E179–82. **(Scientific evidence–III)**
- Morris, C. S., F. B. Rogers, K. E. Najarian, et al. Current trends in vena caval filtration with the introduction of a retrievable filter at a level I trauma center. *J Trauma* 57 (1) (2004): 32–36. **(Scientific evidence–IV)**
- Morse, J. M., and B. Doberneck. Delineating the concept of hope. *Image J Nurs Sch* 27 (4) (1995): 277–85. **(Scientific evidence–NA)**
- Murakami, R., H. Tajima, K. Ichikawa, et al. Acute traumatic injury of the distal descending aorta associated with thoracic spine injury. *Eur Radiol* 8 (1) (1998): 60–62. **(Scientific evidence–IV)**
- Nathens, A. B., J. L. Johnson, J. P. Minei, et al. Inflammation and the host response to injury, a large-scale collaborative project: Patient-Oriented Research Core: Standard operating procedures for clinical care. I. Guidelines for mechanical ventilation of trauma patients. *J Trauma* 59 (3) (2005): 764–69. **(Scientific evidence–Guideline)**
- Nathens, A. B., G. J. Jurkovich, R. V. Maier et al. Relationship between trauma center volume and outcomes. *JAMA* 285 (9) (2001): 1164–71. **(Scientific evidence–III)**
- National SCI Statistical Center. *Spinal Cord Injury: Facts and Figures at a Glance* (June 2006). Available online at www.spinalcord.uab.edu/show.asp?durki=21446. **(Scientific evidence–V)**
- Neighbor, M. L., S. Honner, and M. A. Kohn. Factors affecting emergency department opioid administration to severely injured patients. *Acad Emerg Med* 11 (12) (2004): 1290–96. **(Scientific evidence–IV)**
- Ng, W. P., M. G. Fehlings, B. Cuddy, et al. Surgical treatment for acute spinal cord injury study pilot study #2: Evaluation of protocol for decompressive surgery within 8 hours of injury. *Neurosurg Focus* 6 (1) (1999): E3. **(Scientific evidence–II)**
- Nictora, A., M. Asahina, T. M. Young, et al. Heat-provoked skin vasodilation in innervated and denervated trunk dermatomes in human spinal cord injury. *Spinal Cord* 44 (4) (2006): 222–26. **(Scientific evidence–III)**
- Pagliacci, M. C., M. G. Celani, M. Zampolini, et al. Gruppo Italiano Studio Epidemiologico Mielolesioni. An Italian survey of traumatic spinal cord injury: The Gruppo Italiano Studio Epidemiologico Mielolesioni study. *Arch Phys Med Rehabil* 84 (9) (2003): 1266–75. **(Scientific evidence–I)**
- Papadopoulos, S. M., N. R. Selden, D. J. Quint, et al. Immediate spinal cord decompression for cervical spinal cord injury: Feasibility and outcome. *J Trauma* 52 (2) (2002): 323–32. **(Scientific evidence–III)**
- Pasnoori, V. R., and M. A. Leeser. Use of aminophylline in the treatment of severe symptomatic bradycardia resistant to atropine. *Cardiol Rev* 12 (2) (2004): 65–68. **(Scientific evidence–IV)**
- Patterson, D. R., C. Miller-Perrin, T. R. McCormick, and L. D. Hudson. When life support is questioned early in the care of patients with cervical-level quadriplegia. *N Engl J Med* 328 (7) (1993): 506–09. **(Scientific evidence–NA)**
- Payen, J. F., O. Bru, J. L. Bosson, et al. Assessing pain in critically ill sedated patients by using a behavioral pain scale. *Crit Care Med* 29 (12) (2001): 2258–63. **(Scientific evidence–III)**
- Peacock, W. J., R. D. Shrosbree, and A. G. Key. A review of 450 stab wounds of the spinal cord. *S Afr Med J* 51 (26) (1977): 961–64. **(Scientific evidence–NA)**
- Pelosi, L., J. Lamb, M. Grevitt, et al. Combined monitoring of motor and somatosensory evoked potentials in orthopaedic spinal surgery. *Clin Neurophysiol* 113 (7) (2002): 1082–91. **(Scientific evidence–Prognostic, Level I)**
- Perry, S. D., B. McLellan, W. E. McLroy, et al. The efficacy of head immobilization techniques during simulated vehicle motion. *Spine* 24 (17) (1999): 1839–44. **(Scientific evidence–II)**
- Peterson, W., W. Charlifue, A. Gerhart, and G. Whiteneck. Two methods of weaning persons with quadriplegia from mechanical ventilators. *Paraplegia* 32 (2) (1994): 98–103. **(Scientific evidence–IV)**
- Piatt, J. H., Jr. Detected and overlooked cervical spine injury in comatose victims of trauma: Report from the Pennsylvania Trauma Outcomes Study. *J Neurosurg Spine* 5 (3) (2006): 210–16. **(Scientific evidence–IV)**
- Pitkin, A. D., P. J. Benton, and J. R. Broome. Outcome after treatment of neurological decompression illness is predicted by a published clinical scoring system. *Aviat Space Environ Med* 70 (5) (1999): 517–21. **(Scientific evidence–III)**

- Poonnoose, P. M., G. Ravichandran, and M. R. McClelland. Missed and mismanaged injuries of the spinal cord. *J Trauma* 53 (2) (2002): 314–20. **(Scientific evidence–IV)**
- Prasad, V. S., A. Schwartz, R. Bhutani, et al. Characteristics of injuries to the cervical spine and spinal cord in polytrauma patient population: Experience from a regional trauma unit. *Spinal Cord* 37 (8) (1999): 560–68. **(Scientific evidence–IV)**
- PREPIC Study Group. Eight-year follow-up of patients with permanent vena cava filters in the prevention of pulmonary embolism: The PREPIC (Prevention du Risque d'Embolie Pulmonaire par Interruption Cave) randomized study. *Circulation* 112 (3) (2005): 416–22. **(Scientific evidence–IV)**
- Price, M. J. and I. G. Campbell. Effects of spinal cord lesion level upon thermoregulation during exercise in heat. *Med Sci Sports Exerc* 35 (7) (2003): 1100–07. **(Scientific evidence–II)**
- Qaiyum, M., P. N. Tyrrell, I. W. McCall, and V. N. Cassar-Pullicino. MRI detection of unsuspected vertebral injury in acute spinal trauma: Incidence and significance. *Skeletal Radiol* 30 (6) (2001): 299–304. **(Scientific evidence–I)**
- Quigley, K. J., and H. M. Place. The role of debridement and antibiotics in gunshot wounds to the spine. *J Trauma* 60 (4) (2006): 814–19; discussion 819–20. **(Scientific evidence–IV)**
- Rabinovici, R., P. Ovadia, G. Mathiak, and F. Abdullah. Abdominal injuries associated with lumbar spine fractures in blunt trauma. *Injury* 30 (7) (1999): 471–74. **(Scientific evidence–IV)**
- Raw, D. A., J. K. Beattie, and J. M. Hunter. Anaesthesia for spinal surgery in adults. *Br J Anaesth* 91 (6) (2003): 886–904. **(Scientific evidence–Review)**
- Rizzolo, S. J., A. R. Vaccaro, and J. M. Cotler. Cervical spine trauma. *Spine* 19 (20) (1994): 2288–98. **(Scientific evidence–Review)**
- Rodriguez, D. J., E. C. Benzel, and F. W. Clevenger. The metabolic response to spinal cord injury. *Spinal Cord* 35 (9) (1997): 599–604. **(Scientific evidence–IV)**
- Rogers, F. B., M. D. Cipolle, G. Velmahos, et al. Practice management guidelines for the prevention of venous thromboembolism in trauma patients: The EAST practice management guidelines work group. *J Trauma* 53 (1) (2002): 142–64. **(Scientific evidence–Guideline)**
- Rogers, T., and L. K. Shokes. Extremity fracture management in acute spinal cord injury. *Top Spinal Cord Inj Rehabil* 11 (1) (2005): 40–47. **(Scientific evidence–Guideline)**
- Rosenfeld, J. F., A. R. Vaccaro, T. J. Albert, et al. The benefits of early decompression in cervical spinal cord injury. *Am J Orthop* 27 (1) (1998): 23–28. **(Scientific evidence–Review)**
- Roth, E., G. Davidoff, P. Thomas, et al. A controlled study of neuropsychological deficits in acute spinal cord injury patients. *Paraplegia* 27 (6) (1989): 480–89. **(Scientific evidence–III)**
- Ruiz-Arango, A. F., V. J. Robinson, and G. K. Sharma. Characteristics of patients with cervical spinal injury requiring permanent pacemaker implantation. *Cardiol Rev* 14 (4) (2006): E8–E11. **(Scientific evidence–IV)**
- Saboe, L. A., J. M. Darrah, K. S. Pain, and J. Guthrie. Early predictors of functional independence 2 years after spinal cord injury. *Arch Phys Med Rehabil* 78 (6) (1997): 644–50. **(Scientific evidence–IV)**
- Sackett, D. L., S. S. Richardson, W. S. Rosenberg, W. and Haynes, R. B. Evidence-Based Medicine—*How to practice and teach EBM*. Churchill Livingstone, 2000. **(Scientific evidence–NA)**
- Sala, F., G. Menna, A. Bricolo, and W. Young. Role of glycemia in acute spinal cord injury: data from a rat experimental model and clinical experience. *Ann NY Acad Sci* 890 (1999): 133–54. **(Scientific evidence–IV)**
- Sampalis, J. S., R. Denis, P. Frechette, et al. Direct transport to tertiary trauma centers versus transfer from lower level facilities: Impact on mortality and morbidity among patients with major trauma. *J Trauma* 43 (2) (1997): 288–95; discussion 295–96. **(Scientific evidence–IV)**
- Sampalis, J. S., R. Denis, A. Lavoie, et al. Trauma care regionalization: A process–outcome evaluation. *J Trauma* 46 (4) (1999): 565–79; discussion 579–81. **(Scientific evidence–III)**
- Scalea, T. M., A. Rodriguez, W. C. Chiu, et al. Focused Assessment with Sonography for Trauma (FAST): Results from an international consensus conference. *J Trauma* 46 (3) (1999): 466–72. **(Scientific evidence–Review)**
- Scanlon, C. Ethical concerns in end-of-life care. *Am J Nurs* 103 (1) (2003): 48–55; quiz 56. **(Scientific evidence–NA)**
- Schaefer, D. M., A. E. Flanders, J. L. Osterholm, and B. E. Northrup. Prognostic significance of magnetic resonance imaging in the acute phase of cervical spine injury. *J Neurosurg* 76 (2) (1992): 218–23. **(Scientific evidence–I)**
- Scivoletto, G., B. Morganti, and M. Molinari. Early versus delayed inpatient spinal cord injury rehabilitation: An Italian study. *Arch Phys Med Rehabil* 86 (3) (2005): 512–16. **(Scientific evidence–II)**
- Selden, N. R., D. J. Quint, N. Patel, et al. Emergency magnetic resonance imaging of cervical spinal cord injuries: Clinical correlation and prognosis. *Neurosurgery* 44 (4) (1999): 785–92; discussion 792–93. **(Scientific evidence–IV)**
- Shapiro, A. P., and R. W. Teasell. Behavioural interventions in the rehabilitation of acute v. chronic nonorganic (conversion/factitious) motor disorders. *Br J Psychiatry* 185 (2004): 140–46. **(Scientific evidence–IV)**
- Sharma, S. B., and M. K. Smith. The importance of cultural assessment. *SCI Nurs* 19 (4) (2002): 177–80. **(Scientific evidence–Guideline)**
- Short, D. J., W. S. El Masry, and P. W. Jones. High-dose methylprednisolone in the management of acute spinal cord injury: A systematic review from a clinical perspective. *Spinal Cord* 38 (5) (2000): 273–86. **(Scientific evidence–Review)**
- Siddall, P. J., D. A. Taylor, J. M. McClelland, et al. Pain report and the relationship of pain to physical factors in the first 6 months following spinal cord injury. *Pain* 81 (1–2) (1999): 187–97. **(Scientific evidence–IV)**
- Siddall, P. J., M. J. Cousins, A. Otte, et al. Pregabalin in central neuropathic pain associated with spinal cord injury. *Neurology* 67 (10) (2006): 1792–1800. **(Scientific evidence–I)**
- Simpson, R. K., Jr., B. H. Venger, and R. K. Narayan. Treatment of acute penetrating injuries of the spine: A retrospective analysis. *J Trauma* 29 (1) (1989): 42–46. **(Scientific evidence–IV)**

- Smith, G. C., D. M. Clarke, D. Handrinos, et al. Consultation-liaison psychiatrists' management of somatoform disorders. *Psychosomatics* 41 (6) (2000): 481–89. **(Scientific evidence–IV)**
- Soderstrom, C. A., D. Q. McArdle, T. B. Ducker, and P. R. Militello. The diagnosis of intra-abdominal injury in patients with cervical cord trauma. *J Trauma* 23 (12) (1983): 1061–65. **(Scientific evidence–IV)**
- Speed, J. Behavioral management of conversion disorder: Retrospective study. *Arch Phys Med Rehabil* 77 (2) (1996): 147–54. **(Scientific evidence–IV)**
- Spinal Cord Injury Thromboprophylaxis Investigators. Prevention of venous thromboembolism in the acute treatment phase after spinal cord injury: A randomized, multicenter trial comparing low-dose heparin plus intermittent pneumatic compression with enoxaparin. *J Trauma* 54 (6) (2003): 1116–24; discussion 1125–26. **(Scientific evidence–I)**
- Spivak, J. M., M. A. Weiss, J. M. Cotler, and M. Call. Cervical spine injuries in patients 65 and older. *Spine* 19 (20) (1994): 2302–06. **(Scientific evidence–II)**
- Stambolis, V., S. Brady, D. Klos, et al. The effects of cervical bracing upon swallowing in young, normal, healthy volunteers. *Dysphagia* 18 (1) (2003): 39–45. **(Scientific evidence–I)**
- Steeves, J., J. Fawcett, and M. Tuszynski. Report of international clinical trials workshop on spinal cord injury, February 20–21, 2004, Vancouver, Canada. *Spinal Cord* 42 (10) (2004): 591–97. **(Scientific evidence–NA)**
- Stevens, S., A. Bhardwaj, J. Kirsch, M. Mirski. Critical care and perioperative management in traumatic spinal cord injury. *J Neurosurg Anesthesiol* 15 (3) (2003): 215–29 **(Scientific evidence–Review)**
- Stroebe, M., and H. Schut. The dual process model of coping with bereavement: Rationale and description. *Death Stud* 23 (3) (1999): 197–224. **(Scientific evidence–NA)**
- Sturm, J. T., J. T. Hynes, and J. F. Perry, Jr. Thoracic spinal fractures and aortic rupture: A significant and fatal association. *Ann Thorac Surg* 50 (6) (1990): 931–33. **(Scientific evidence–II)**
- Sullivan, J. Surviving uncertainty and projecting recovery: A qualitative study of patients' and family members' experiences with acute spinal cord injury. *SCI Nurs* 18 (2) (2001): 78–86. **(Scientific evidence–IV)**
- Sung, J., G. V. Bochicchio, M. Joshi, et al. Admission hyperglycemia is predictive of outcome in critically ill trauma patients. *J Trauma* 59 (1) (2005): 80–83. **(Scientific evidence–IV)**
- Taneichi, H., K. Suda, T. Kajino, and K. Kaneda. Traumatically induced vertebral artery occlusion associated with cervical spine injuries: Prospective study using magnetic resonance angiography. *Spine* 30 (17) (2005): 1955–62. **(Scientific evidence–IV)**
- Tator, C. H. Early management of acute spinal cord injury. In *Seminars in Neurological Surgery*, ed. C. H. Tator. New York: Raven Press, 1982. **(Scientific evidence–Guideline)**
- Tator, C. H. Acute management of spinal cord injury. *Br J Surg* 77 (5) (1990): 485–86. **(Scientific evidence–Guideline)**
- Tator, C. H., E. G. Duncan, V. E. Edmonds, et al. Complications and costs of management of acute spinal cord injury. *Paraplegia* 31 (11) (1993): 700–14. **(Scientific evidence–III)**
- Tator, C. H., M. G. Fehlings, K. Thorpe, and W. Taylor. Current use and timing of spinal surgery for management of acute spinal surgery for management of acute spinal cord injury in North America: Results of a retrospective multicenter study. *J Neurosurg* 91 (1 Suppl) (1999): 12–18. **(Scientific evidence–IV)**
- Taylor, S. E., and J. D. Brown. Positive illusions and well-being revisited: Separating fact from fiction. *Psychol Bull* 116 (1) (1994): 21–27; discussion 28. **(Scientific evidence–Review)**
- Teasell, R. W., and A. P. Shapiro. Strategic-behavioral intervention in the treatment of chronic nonorganic motor disorders. *Am J Phys Med Rehabil* 73 (1) (1994): 44–50. **(Scientific evidence–IV)**
- Thorell, A., O. Rooyackers, P. Myrenfors, et al. Intensive insulin treatment in critically ill trauma patients normalizes glucose by reducing endogenous glucose production. *J Clin Endocrinol Metab* 89 (11) (2004): 5382–86. **(Scientific evidence–III)**
- Tisherman, S. A., P. Barie, F. Bokhari, et al. Clinical practice guideline: Endpoints of resuscitation. *J Trauma* 57 (4) (2004): 898–912. **(Scientific evidence–Guideline)**
- Torina, P. J., A. E. Flanders, J. A. Carrino, et al. Incidence of vertebral artery thrombosis in cervical spine trauma: Correlation with severity of spinal cord injury. *AJNR Am J Neuroradiol* 26 (10) (2005): 2645–51. **(Scientific evidence–III)**
- Toscano, J. Prevention of neurological deterioration before admission to a spinal cord injury unit. *Paraplegia* 26 (3) (1988): 143–50. **(Scientific evidence–Guideline)**
- Transportation of patients with acute traumatic cervical spine injuries. *Neurosurgery* 50 (3 Suppl) (2002): S18–20. **(Scientific evidence–Guideline)**[[Not cited in the text.]]
- Trieschmann, R. *Spinal Cord Injuries: Psychological, Social, and Vocational Rehabilitation*. New York: Demos Publication, 1988. **(Scientific evidence–NA)**
- Tsirikos, A. I., J. Aderinto, S. K. Tucker, and H. H. Noordeen. Spinal cord monitoring using intraoperative somatosensory-evoked potentials for spinal trauma. *J Spinal Disord Tech* 17 (5) (2004): 385–94. **(Scientific evidence–IV)**
- U. S. Department of Health and Human Services Program Support Center, Visual Communications Branch. *Model Trauma System Planning and Evaluation*. Rockville, MD: Health Resources and Services Administration, 2006. **(Scientific evidence–Guideline)**
- U. S. Department of Health and Human Services Program Support Center, Visual Communications Branch. *Resources for Optimal Care of the Injured Patient*. Rockville, MD: Health Resources and Services Administration, 2006b. **(Scientific evidence–Guideline)**
- Vaccaro, A. R., H. S. An, S. Lin et al. Noncontiguous injuries of the spine. *J Spinal Disord* 5 (3) (1992): 320–29. **(Scientific evidence–IV)**
- Vaccaro, A. R., R. J. Daugherty, T. P. Sheehan, et al. Neurologic outcome of early versus late surgery for cervical spinal cord injury. *Spine* 22 (22) (1997): 2609–13. **(Scientific evidence–II)**
- Vaccaro, A. R., S. P. Falatyn, A. E. Flanders, et al. Magnetic resonance evaluation of the intervertebral disc, spinal ligaments, and spinal cord before and after closed traction reduction of cervical spine dislocations. *Spine* 24 (12) (1999): 1210–17. **(Scientific evidence–IV)**

- Vale, F. L., J. Burns, A. B. Jackson, and M. N. Hadley. Combined medical and surgical treatment after acute spinal cord injury: Results of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg* 87 (2) (1997): 239–46. **(Scientific evidence–IV)**
- Van den Berghe, G., A. Wilmer, G. Hermans, et al. Intensive insulin therapy in the medical ICU. *N Engl J Med* 354 (5) (2006): 449–61. **(Scientific evidence–I)**
- Van den Berghe, G., P. Wouters, F. Weekers, et al. Intensive insulin therapy in critically ill patients. *N Engl J Med* 345 (19) (2001): 1359–67. **(Scientific evidence–I)**
- Velmahos, G. C., K. Toutouzas, L. Chan, et al. Intubation after cervical spinal cord injury: To be done selectively or routinely? *Am Surg* 69 (10) (2003): 891–94. **(Scientific evidence–IV)**
- Vickery, D. The use of the spinal board after the prehospital phase of trauma management. *Emerg Med J* 18 (1) (2001): 51–54. **(Scientific evidence–Review)**
- Vioreanu, M. H., J. F. Quinlan, I. Robertson, and J. M. O’Byrne. Vertebral fractures and concomitant fractures of the sternum. *Int Orthop* 29 (6) (2005): 339–42. **(Scientific evidence–IV)**
- Vogelzang, M., J. M. Nijboer, I. C. van der Horst, et al. Hyperglycemia has a stronger relation with outcome in trauma patients than in other critically ill patients. *J Trauma* 60 (4) (2006): 873–77; discussion 878–79. **(Scientific evidence–III)**
- Wagner, R. L., P. F. White, P. B. Kan, et al. Inhibition of adrenal steroidogenesis by the anesthetic etomidate. *N Engl J Med* 10 (22) (1984): 1415–21. **(Scientific evidence–IV)**
- Walters, K., and J. R. Silver. Gastrointestinal bleeding in patients with acute spinal injuries. *Int Rehabil Med* 8 (1) (1986): 44–47. **(Scientific evidence–IV)**
- Wang, C. M., Y. Chen, M. J. DeVivo, and C. T. Huang. Epidemiology of extraspinal fractures associated with acute spinal cord injury. *Spinal Cord* 39 (11) (2001): 589–94. **(Scientific evidence–II)**
- Waters, R. L., I. Sie, R. H. Adkins, and J. S. Yakura. Motor recovery following spinal cord injury caused by stab wounds: A multicenter study. *Paraplegia* 33 (2) (1995): 98–101. **(Scientific evidence–IV)**
- Webborn, N., M. J. Price, P. C. Castle, et al. Effects of two cooling strategies on thermoregulatory responses of tetraplegic athletes during repeated intermittent exercise in the heat. *J Appl Physiol* 98 (6) (2005): 2101–07. **(Scientific evidence–I)**
- Welage, L. S. Overview of pharmacologic agents for acid suppression in critically ill patients. *Am J Health Syst Pharm* 62 (10 Suppl 2) (2005): S4–S10. **(Scientific evidence–NA)**
- Wiberg, J., and H. N. Hauge. Neurologic outcome after surgery for thoracic and lumbar spine injuries. *Acta Neurochir* 91 (3–4) (1988): 106–12. **(Scientific evidence–IV)**
- Wilberger, J. E. Immobilization and traction. In *Contemporary Management of Spinal Cord Injury: From Impact to Rehabilitation*, eds. C. H. Tator and E. C. Benzel. Park Ridge, IL: American Association of Neurological Surgeons, 2000: 91. **(Scientific evidence–NA)**
- Wilson, J. T., F. B. Rogers, S. L. Wald, et al. Prophylactic vena cava filter insertion in patients with traumatic spinal cord injury: Preliminary results. *Neurosurgery* 35 (2) (1994): 234–39; discussion 239. **(Scientific evidence–III)**
- Winemiller, M. H., K. A. Stolp-Smith, M. D. Silverstein, and T. M. Therneau. Prevention of venous thromboembolism in patients with spinal cord injury: Effects of sequential pneumatic compression and heparin. *J Spinal Cord Med* 22 (3) (1999): 182–91. **(Scientific evidence–II)**
- Wing, P. C., P. Nance, D. G. Connell, and F. Gagnon. Risk of avascular necrosis following short-term megadose methylprednisolone treatment. *Spinal Cord* 36 (9) (1998): 633–36. **(Scientific evidence–III)**
- Winslow, C., R. K. Bode, D. Felton et al. Impact of respiratory complications on length of stay and hospital costs in acute cervical spine injury. *Chest* 121 (5) (2002): 1548–54. **(Scientific evidence–II)**
- Winslow, J. E., 3rd, R. Hensberry, W. P. Bozeman et al. Risk of thoracolumbar fractures doubled in victims of motor vehicle collisions with cervical spine fractures. *J Trauma* 61 (3) (2006): 686–87. **(Scientific evidence–IV)**
- Wolf, C., and T. H. Meiners. Dysphagia in patients with acute cervical spinal cord injury. *Spinal Cord* 41 (6) (2003): 347–53. **(Scientific evidence–II)**
- Wortman, C. B. and R. C. Sivler. The myths of coping with loss. *J Consult Clin Psychol* 57 (3) (1989): 349–57. **(Scientific evidence–N/A)**
- Wyte, S. R. Ketamine-induced intracranial hypertension. *Anesthesiology* 36 (2) (1972): 174–76. **(Scientific evidence–IV)**
- Yarkony, G. M., L. M. Bass, V. Keenan 3rd, and P. R. Meyer Jr. Contractures complicating spinal cord injury: Incidence and comparison between spinal cord centre and general hospital acute care. *Paraplegia* 23 (5) (1985): 265–71. **(Scientific evidence–II)**
- Yendamuri, S., G. J. Fulda, and G. H. Tinkoff. Admission hyperglycemia as a prognostic indicator in trauma. *J Trauma* 55 (1) (2003): 33–38. **(Scientific evidence–II)**
- Yoo, K. Y., S. W. Jeong, S. J. Kim et al. Cardiovascular responses to endotracheal intubation in patients with acute and chronic spinal cord injuries. *Anesth Analg* 97 (4) (2003): 1162–67, table of contents. **(Scientific evidence–III)**
- Yugue, I., K. Shiba, T. Ueta, and Y. Iwamoto. A new clinical evaluation for hysterical paralysis. *Spine* 29 (17) (2004): 1910–13; discussion 1913. **(Scientific evidence–I)**